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Editorials

WHOSE HOSPITALIZATION PROGRAM?

The direction of political interest in the last two months has tended toward war and reelection with the latter item probably being a most consuming one. When reelection is "sure" political action in the legislative halls is fast but when elections are hard fought and close our representatives feel the sting of criticism and are more inclined to be deliberate and temperate in their manipulations. In an atmosphere of "sureness" and of cock-sure rightness much of our social legislation in the last few years has been proposed and passed. The "rightness" of any proposal is of course its tendency to assist in the reelection of its sponsor. Now that elective campaigns are in full swing and war issues obtrude themselves there may be for a time a dull in social proposals. While this quiet exists there may be time to examine some of the motivations back of the latest "health" proposal—the Wagner-George-Lea bill for the federal construction of hospitals.

This Senate bill was apparently formulated at the behest of the President who undoubtedly acted at the urging of certain advisors. These socially-minded individuals have been remarked on in these columns before—sufficient to say that they are not physicians acquainted with the practice of medicine. According to an editorial in the January 20, 1940, issue of the *Journal of the American Medical Association* a memorandum left with the President by a group of competent medical advisors suggested certain objectives to which the hospital building program should conform. Remarks of Senator Wagner at a hearing of the bill on March 18 indicated that in his mind and according to his information a certain type of hospital was necessary in the "needy" areas. This hospital, also recommended by the President's medical committee, is commonly known as a general hospital. Now this is important to bear in mind for several reasons. First of all it suggests that the hospital building program is outside the field of the Public Health

Service. And of course it has no relation to the general hospitalization procedures of the Veterans Facility. So this hospital financing maneuver is to land the federal government into a new field in caring for the sick. True, the proposed bill does not contemplate it; it expects the county and municipal agencies in impoverished areas to finance the operation of the venture. But it is submitted here that a community too poor to bond itself to build the hospital will hardly be able to continuously meet the much larger burden of operating the institution. And thus the federal government will find itself in the general hospital business along with its farming, hotel operation and sundry other occupations demonstrative of the combined wishful thinking and bad business judgment of powerful political pressure groups.

It is contemplated that the hospitals erected under the federal program will have a capacity of from 50 to 100 beds. The proponents of this particular idea likely do not know something immediately apparent to any practitioner in the rural districts under consideration—that 50 to 100 bed hospitals, if they were to be filled, would be as far away as the now available hospitals. There are in Illinois a number of very small hospitals in rural communities meeting the local emergency needs. The need for such institutions in Colorado was suggested by Dr. R. L. Cleere in a letter introduced before the hearings by Dr. Parran. Other letters from state health officers were introduced by Dr. Parran. These health officers stuck pretty much to their business; almost all of them could use in their respective states more accommodations for tuberculous patients or for contagious diseases. There apparently was not much pressure in the various states for hospitals for the management of general diseases. So one wonders if the hospitalization program isn't Dr. Parran's program and if he is trying to project the public health service into general practice. In our own state Dr. A. C. Baxter described in his letter the lack of accommodations for tuberculous patients in the southern part of the state but he also mentioned that there was enabling legislation allowing counties to group together for constructing sanatoria.

Certainly one of the glaring faults in the hospital bill is that it seems to have been approached from the public health angle and at a distance from its intended beneficiaries. It has been suggested that the techniques and procedures of public health may not, when applied to hospital management, give proper consideration to "local differences." Amidst all the differences in varying localities where hospitalization may be remote or inadequate—poor agriculture, sparse range, played-out mines—there is one common interest and that is good transportation. Should the social thinkers see their clear call they would urge the President, or Dr. Parran or perhaps the appropriate department to extend good roads and keep them open. Then if they watched they would observe the physician make his easy though distant rounds and the needful sick would be transported to a substantial and properly equipped hospital. Decidedly all this goes on now though handicapped at times by mud and snow. As the last twenty years have seen an extension of the facilities for ordinary travel they have seen an improvement in medical service. The second is not exclusively dependent on the first for many changes have occurred and are still going on, the benefits of science being applied shortly after they are learned all over this country. Perhaps the best expression of the practitioner's feeling was given by Rev. Alphonse M. Schwitalla before the 1939 meeting of the American Hospital Association as part of a talk on "The Place of Government Hospitals in the General Hospital Field," he said, "Perhaps it has not been sufficiently pointed out that one of the causes for resentment against the present program of governmental control lies precisely in the fact that the inter-departmental committee attempted through governmental coercion to change a spontaneous and naturally growing trend into a directed and coercively planned evolutionary process."

Again we ask "Whose Hospitalization Program Is This?" Does it belong to the Public Health Service or to the inter-departmental committee mentioned by Rev. Schwitalla? It answers no problems of the practitioner and does not meet the needs of the people.

THE NEW YORK MEETING OF THE AMERICAN MEDICAL ASSOCIATION

The American Medical Association held its ninety-first annual session in New York, June 10 to 14.

New York is to be congratulated on having been host to such a successful convention of this, the largest medical organization in the world. There was a registration of 12,864 out of a mem-

bership of approximately 117,000. Registration in recent meetings have been: 1939, St. Louis, 7,412; 1938, San Francisco, 6,000; 1937, Atlantic City, 9,764; 1936, Kansas City, 6,824; 1935, Atlantic City, 8,166. Naturally, a large attendance expected when the organization meets in the centers of medical population, as was the case this time. However, and no doubt about it, the fact that the World's Fair was held over for this year was the added inducement. The attendance from Illinois was 500.

The Illinois State Medical Society was represented in the House of Delegates by its full quota of nine members as follows: Charles B. Reed, Chicago; W. E. Kittler, Rochelle; C. E. Wilkinson, Danville; L. O. Frech, Decatur; Charles J. Whalen, Chicago; John J. Pflock, Chicago; G. Henry Mundt, Chicago; Rollo K. Packard, Chicago; Edwin S. Hamilton, Kankakee; Harold M. Camp, Monmouth, who represented the State Society in a secretarial capacity.

The transactions of the House of Delegates was marked by harmony and expedition. Reference committee reports were considered in a serious and earnest fashion and with few exceptions harmoniously and expeditiously dispatched.

The address of the speaker was a constructive elaboration on the activities of the organization during the past year. Homage was paid to the memory of those who died since the 1939 meeting of the organization.

Reviewing the work of the association during the past year and suggesting some of the problems which may arise in the near future, Dr. Sleyster addressing the House of Delegates, recalled the problems which have constantly faced the organization in recent years and declared that "The American Medical Association stands almost alone in defense of an historic system of freedom of science and its application for human welfare—a system which shows with pride a record unrivaled in the benefits it has brought to humanity."

"At this session," he concluded, "let us rededicate ourselves to a defense of those principles and policies so necessary to preserve the accomplishments of the past, so necessary to guarantee progress in the future."

Dr. Van Etten as president-elect in his address referred to present dictatorial tendencies throughout the world and to the present threat of aggression as a part of a world-wide plan to destroy the democracies. He dwelt with considerable emphasis on present legislative endeavors in Washington and cited the Hospital Construction Bill, the Wagner-George Bill now before Congress and mentioned by way of emphasis that statements had been recently made in Washington that the Hospital Construction Bill is considered the first step in a comprehensive federal health plan, and that it will lead to other steps in that direction in the near future.

War emergency sentiment was reflected in four resolutions introduced into the house of delegates, the association's democratic policy making "medical parliament," representing 117,000 American physicians.

The House adopted a resolution calling for unwavering support of the President of the United States in his stand on the need for national unity and organization in any possible emergency. The delegates admitted the necessity of surrendering a measure of freedom under military necessity, but contend that freedom should be restored after the emergency has passed.

A resolution for medical mobilization was adopted, approving of a tentative plan for the procurement of professional personnel for the medical corps of the U. S. Army. The plan was prepared in the office of the Surgeon General of the Army and presented to the house of delegates by the military delegate from the Army. We quote the resolution:

1. The American Medical Association to be asked to conduct a survey of the medical profession through its state and local societies.

2. The local or county societies to canvass their members to determine of those who express a willingness to serve, who should be available for the military service and who, on account of their age, physical disability, or commitment in civil capacities should remain at home.

3. The county society to give to each one who expresses his willingness to serve, even though he may be selected to remain at home, a button similar to that which was designed for the Volunteer Medical Service Corps during the last war.

4. The county societies to list those who are selected for the military service according to their professional qualifications, listing as surgeons, psychiatrists, etc., only those who are members in the national specialists' organizations. Also, to select from those who are to remain at home, qualified men for examination boards.

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5. The state societies to maintain an available roster of their members.

6. The American Medical Association to maintain a numerical roster of availability by states.

7. The Medical Departments of the Army to have one or more selected officers on duty at headquarters of A.M.A. in Chicago.

8. The War Department, Corps Areas, or regional officers to call upon the A.M.A. for physicians or specialists, as and when required.

9. The American Medical Association to call upon the states, according to their quotas, for the physicians required.

10. The state, in turn, to call upon its local societies for its quota of physicians.

"In the quotas, credits would be given for sponsored units, and preference would be given to reserve officers wherever their qualifications warrant.

AVOIDS COMPLETE LOSS

"It appears that in the event of a national emergency of great magnitude that it would be very necessary to conserve the medical profession. This plan would distribute the professional load and, if properly administered, should prevent the stripping of rural and isolated communities of their necessary medical personnel.

"There could be an extension of this plan to cover the training program for technicians. The same societies could conduct a survey of the teaching institutions to determine their availability and suitability for the training of such enlisted specialists as would be required. Rational medical service for civilian groups in war industries could be coordinated by the same administrative units"

A resolution on medical preparedness introduced by the chairman of the board of trustees as follows:

WHEREAS, The ravages of war again pervade many of the nations and peoples of the world; and

WHEREAS, The President of the United States has indicated to the nation and to the Congress the desirability of military preparedness so that our people may successfully resist attempts to substitute other forms of government for the democracy established by the constitution of our country; and

WHEREAS, Organization of the nation for preparedness involves from the first the complete cooperation of the physicians of the country for

1. Medical services in the Military, Naval, Aviation, and Veteran's administrations;

2. Selection of men physically fit to serve with such agencies; and

3. Rehabilitation of those not physically qualified to participate in military activities; and

WHEREAS, Preparedness demands also

1. Medical service to the industrial workers engaged in war industries;

2. Continuance of medical care of the civilian population;

3. Education of young men to qualify them for medical service; and

WHEREAS, The American Medical Association now embraces in its membership more than 117,000 of the licensed physicians of the United States; and

WHEREAS, The headquarters facilities of the American Medical Association has available.

1. Complete records of all qualified physicians in this country, with data necessary to determine largely their availability for military or other services;

2. Complete information concerning facilities for education in medicine, the medical specialties, and other medical activities;

3. Complete information concerning the hospitals for the United States;

4. The necessary facilities for making prompt contact through addressing devices periodicals, and constituent bodies with all medical personnel and medical agencies; and

WHEREAS, Only in the headquarters of the American Medical Association, as far as is known, are such information and facilities available; and

WHEREAS, The American Association is not only the largest but also the only organization containing in its membership qualified physicians in every field of medical practice; and

WHEREAS, During the World War of 1914-1918 the American Medical Association aided in making available the services of more than 60,000 physicians for military and related activities: therefore be it

RESOLVED, That the House of Delegates authorize the Board of Trustees to create a Committee on Medical Preparedness, to consist of seven members of this House, with the President of the Association, the Secretary of the Association, the Secretary of the Board of Trustees, and the Editor as ex officio members; and be it further

RESOLVED, That this Committee establish and maintain contact and suitable relationship with all governmental agencies concerned with the prevention of disease and care of the sick, in both civil and military aspects, so as to make available at the earliest possible moment every facility that the American Medical Association can offer for the health and safety of the American people and the maintenance of American democracy.

A resolution was introduced in the interest of the American Medical Women's Association, calling upon the Government to extend recognition to women physicians in the form of commissions and ratings, as in the case of male physicians.

A resolution on the care of needy physicians was as follows:

WHEREAS, The care of physicians who are in need is a subject worthy of the attention of the constituted units of organized medicine; and

WHEREAS, a serious study and survey of this problem is definitely indicated; now be it

RESOLVED, By this House of Delegates of the American Medical Association that the speaker of the house of delegates be instructed to appoint a committee of three to make a study of the problem of (1) aid to needy members of the association, and, (2) establishment of a national fund for this purpose, and to submit a report of this study with recommendations at the next annual session of the house of delegates.

A resolution from Mississippi cited the fact that Senator Bilbo of that State, had introduced into the senate of the United States, a resolution declaring June 22nd as "Doctors' Day," in recognition for the services rendered by the medical profession to the people. A resolution from Alabama, called for the closest co-operation between the medical profession and the health authorities in the effort to control venereal diseases, and recommending that physicians who serve in the various venereal disease clinics be compensated for their services.

A resolution calling upon constituent State Associations and component County Medical societies, to work out plans whereby some protection would be given the practice of physicians called into the service, and perhaps some remuneration therefrom.

A request was received from the National Medical Association, the national organization of Negro physicians, that negro physicians be included in any plans set up for the procurement of physicians for the armed forces in the anticipated emergency.

Another matter which received careful consideration at the hands of both the house and of its reference committee on legislation and public relations was the Federal Hospital Construction Bill which had been radically altered in the senate committee to which it had been referred and had passed the senate. While approval was given to the broad principles embodied into the bill, several suggestions were offered as to how the bill might be improved. Strong objections were interposed, to a last minute amendment written into the bill by Senator Murray from the floor, which included the Osteopathic profession as one of the groups from which the eight members of the national advisory council may be selected.

The House rejected a plea from Louisiana to endorse repeal of some of the restrictions placed on heroin by the narcotic law. (The A.M.A. has long held that heroin is not indispensable as a medicine.)

The exhibits, both scientific and technical, were most extensive and interesting; and, for the many who concentrated upon them, proved a valuable asset in stimulating a thirst for newer, scientific knowledge.

Two hundred and fifty papers were scheduled for delivery before the various general and scientific sections, many of them illustrated by scientific exhibits which numbered nearly two hundred and fifty, covering the entire floor of the Grand Central Palace.

Every third year in accordance with the provision of the by-laws a reapportionment of delegates has to be made. This was done at the 1940 session on a basis of one delegate from each state for every 930 members or a fraction thereof. The total membership of the House, including representation from the three federal services and the various sections is limited to 175, also at this session a new section on Anesthesiology was created, making the total number of sections now, sixteen.

This reapportionment resulted in a gain of one delegate for four states, with a corresponding loss in four other states. Illinois' present allotment of nine delegates was not altered.

Three names were submitted by the Board of Trustees as candidates for the distinguished service medal and citation. From the names

submitted one candidate could be elected. The honor was bestowed upon Dr. Chevalier Jackson of Philadelphia, now 74 years of age and whose outstanding contribution in the field of bronchoscopy are known wherever scientific medicine is practiced.

Without opposition, the House selected as president-elect Dr. Frank H. Lahey, of Boston, who has long been identified with the association's activities in the field of graduate education.

Dr. W. F. Braasch, of Rochester, Minnesota, was elected a member of the Board of Trustees to fill the vacancy created by the recent untimely death of Dr. Charles B. Wright of Minneapolis. No other changes were made in the Board of Trustees.

PRIORITY OF USE OF LUNG COLLAPSE THERAPY, SUBSEQUENT RECORD BROUGHT UP TO DATE

In the "Epitomized Record of Progress of Medicine During the Last Hundred Years," (ILLINOIS MEDICAL JOURNAL, June, 1940) the important subject of collapse therapy did not receive sufficient attention. The following paragraphs are designed to remedy the oversight.

Prior to its actual application by Forlanini, collapse therapy had been suggested or foreshadowed by many writers. In 1692 Purnamm advocated incision of the chest wall in hemorrhage. In 1710, Boglinus advised incision of the pleura for treatment of phthisical cavities. In 1726, Berry of Dublin, suggested drainage of pulmonary cavities. In 1769, Sharpe, trail breaking for Jacobaeus, discussed the separation of pleuritic adhesions. In 1821, Carson clearly recognized the potentialities of pulmonary collapse and proved the practicability of his doctrine by animal experimentation. In 1884, Potain injected sterilized air to replace a spontaneous hydro-pneumothorax.

In 1888, Forlanini actually began his pneumothorax work and reported his cases in 1894 and 1895. Dr. John B. Murphy, according to some of his colleagues, seems to have questioned the priority of Forlanini. In 1898, at the Dunning Tuberculosis Institute, Murphy and Tice performed pneumothorax on ambulant cases. Prior to this Murphy had undoubtedly done some pneumothorax work and reported five cases in 1898.

About 1902, interest in pneumothorax commenced to fade. A few workers as Von Ruck in this country, Brauer, Adolph Schmidt, Forlanini, Dumarest, Arcelin and Kuss in Europe, maintained their faith in the procedure, and continued the treatment. Around 1912 there came a gradual revival of interest and in this country amongst the pioneers of the era were Mary Lapham, Gerald B. Webb of Colorado Springs, Rothschild of San Francisco, Von Ruck and a few others. Amongst workers of a slightly later date may be mentioned the Matsons, Coryllos, O'Brien, Hedblom, Pollock and others.

Until 1931, however, collapse therapy had been recognized largely as a hospital procedure. In that year (1931) impelled by a shortage of beds at the Municipal Tuberculosis Sanitarium, Chicago, Tice and Hruby introduced field collapse in widespread application for patients who could not gain admission to institutions. This program of extra-mural pneumothorax, including treatment from the initial injection onward, has gradually expanded until, as of January 1, 1940, there were 1,861 cases under supervision, 1,290 cases actually under treatment, and 334 postoperative cases under observation. The entire four floors of the old Iroquois Hospital, equipped with a few beds for emergencies, are being used for ambulant cases. It is of interest to know that this is the first hospital devoted exclusively to collapse therapy.

ALIEN REGISTRATION IS COMPULSORY

Alien Registration Division of Immigration and Naturalization Service, Department of Justice, has asked us to publish the following:

As part of the National Defense program, a nationwide registration of aliens will be conducted from August 27 through December 26, 1940, by the Immigration and Naturalization Service of the Department of Justice. Registration will take place in the post offices of the nation. It is expected that more than three and one-half million aliens will be registered during the four-month period.

Registration is made compulsory by a specific act of Congress, the Alien Registration Act of 1940, which requires all noncitizens to register during the four-month official registration period. The law requires that all aliens 14 years or older are to be registered and fingerprinted. Alien children under 14 years of age will be registered by

their parents or guardians. When alien children reach their fourteenth birthday, they will be required to register in person and be fingerprinted.

A fine of $1,000 and imprisonment of six months is prescribed by the Alien Registration Act for failure to register, for refusal to be fingerprinted, or for making registation statements known to be false.

As part of its educational program to acquaint non-citizens with the registration requirements, the Alien Registration Division is distributing more than five million specimen forms listing the questions that will be asked of aliens at registration time. Besides the usual questions for establishing identification, the questionnaire asks the alien to tell how and when he entered the country, the method of transportation he used to get here, the name of the vessel on which he arrived.

He is also asked to state the length of time he has been in this country and the length of time he expects to stay. He must also describe any military or naval service he has had, and list the names of any organizations, clubs, or societies in which he participates or holds membership. In addition, he is required to describe his activities in any organization, and to affirm whether or not the organization furthers the interests or program of a foreign government.

To make their registration easier, aliens are being asked to fill out sample forms, which will be available prior to registration, and take them to post offices where they will be registered and fingerprinted. Every registered alien will receive by mail a receipt card which serves as evidence of his registration. Following registration, the Act requires all aliens, as well as parents or guardians of alien children, to report changes of residence address within five days of the change.

The Alien Registration Act was passed so that the United States Government may determine exactly how many aliens there are, who they are, and where they are. Both President Roosevelt and Solicitor General Biddle have pointed out that registration and fingerprinting will not be harmful to law-abiding aliens. The Act provides that all records be kept secret and confidential. They will be available only to persons approved by the Attorney General of the United States.

Fingerprinting of aliens carries no stigma whatsoever. Thousands of citizens are voluntarily fingerprinted every year. Members of the United States Army and Navy are all fingerprinted, as are many Government workers. In recent years, many hospitals have established the practice of taking footprints of newly-born babies. Because fingerprinting is the only infallible method of accurate identification, the United States Government has adopted is at part of its registration program.

In signing the Alien Registration Act, President Roosevelt said, "The Alien Registration Act of 1940 . . . should be interpreted and administered as a program designed not only for the protection of the country but also for the protection of the loyal aliens who are its guests. The registration . . . does not carry with it any stigma or implication of hostility towards those who, while they may not be citizens, are loyal to this country and its institutions. Most of the aliens in this country are people who came here because they believed and had faith in the principles of American democracy, and they are entitled to and must receive full protection of the law."

Solicitor General Biddle adds, "We should remember that all Americans were at one time or another immigrants from other lands. The genius of many countries, the ancient aspirations of many races, have built into what is America. Unfortunately, there are some foreigners who are disloyal to America, who do not wish to accept our ways and who use our freedom of speech and of the press to foment disunity and sedition.

"These persons we will apprehend, but we will also see to it that loyal American aliens are not unjustly condemned for the disloyal behavior of a few. Our registration will be their protection from persecution."

The Immigration and Naturalization Service asks for the cooperation of all citizens in carrying out the Alien Registration program in a friendly manner so that our large foreign population is not antagonized. It is suggested that citizens may be of great help to their non-citizen neighbors or relatives by explaining to those who do not speak English well what the registration is, where aliens go to register, and what information they must give.

The Registration of Aliens program has been set up as a separate division of the Immigration and Naturalization Service. The program is being directed by Earl G. Harrison, under the general supervision of Major L. B. Schofield, Special Assistant to the Attorney General.

MEDICAL ECONOMICS

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Careful attention to the platforms of both parties as well as the talks broadcast by radio and printed in the daily press, might lead one to believe that the attention of the reformers was no longer directed toward such minor things as reforming the practice of medicine. Aside from a brief reference by Senator Wagner in one of the preambles to the adopted platform of the Democratic platform, of their interest in obtaining better medical care for the nation, there was no mention of either the need or the imminence of the socialization of medicine. However, the writer refuses to believe that the above is true but is rather inclined to think that such a minor reformation has been temporarily pigeon-holed so that some of the more prolific vote producing subjects can temporarily be presented to the public in an effort to win the election in November. But at least it is another breathing spell, for apparently the present Congress will be very busy on highly controversial subjects the rest of the time it is in session. There remains the ever present danger of some plan being rushed through either by being included in some other bill to be passed, or by some adroit politician grasping an opportunity of putting through his pet bill, while many of opponents are away trying to be reelected. Constant vigilance in Washington is still necessary.

After reading the two most excellent advertisements in the issue of *Saturday Evening Post* immediately preceding both the Democrat and the Republican conventions, under the sponsorship of the National Committee for the Extension of Medical Service, and then asking the readers of the magazine for their reaction, the writer was astonished to find that little if any impression was made on the reading public and certainly little on the delegates. If the reaction of readers of articles of propaganda presented so frequently in the lay press is no better than in the above instance, one must wonder whether some of us have been too alarmed by the influence of the flood of propaganda against the medical profession in the past three years in the lay press. Certainly the futility of the medical profession attempting to compete with the paid propagandists of the reformers is made more apparent than ever.

The death of Dr. A. A. Hayden of Chicago a few weeks ago is a definite and distinct loss to the medical profession of Illinois. He has been intensely interested in medical economic affairs and has been of great assistance to this committee on many occasions. We wish to show our appreciation at this time, for his many favors.

Apparently there is some misinformation as to the status of the suit of the United States Government against the American Medical Association and some of its officers. The Supreme Court has not declared the practice of medicine a trade as erroneously reported in a current periodical. Rather it has agreed with the decision of the Court of Appeals that the case should be returned to the District Court of the United States of the District of Columbia for trial. The defendants in this suit have filed pleas of not guilty and the time of the trial has not as yet been set. Until this trial has been held and a decision as to guilt made, this case cannot be decided by either the Court of Appeals or the Supreme Court of the United States. Those of you, who are interested in this case, and every man practicing medicine in the State of Illinois should be, can get all desired information in the *Journal of the AMA*, page 222 of the issue of July 20, 1940. On page 224 of the same issue, is a report of the results of a years trial of Farm Security Administrations plan for furnishing medical service to their clients in Avoyelles Parish, Louisiana. Another group of physicians have tried out the plan and find it unsatisfactory. We will refrain from any personal comment at this time, for the view

of the writer is only too well known on this subject to the readers of the same.

The California Physicians Service, formed to furnish Voluntary health insurance to groups in California has been obliged to alter its policy, due to the fact that one years operation has netted only 12,000 members.

This is all together too few members to make the plan successful and there is an attempt to make the plan more inclusive by either lessening the cost or changing the classification of eligibles. We watch the experiment of the Michigan Medical Society in this field with great interest because it is nearby and we were present at the meeting of the Society when the plan was decided on. At the last meeting of the House of Delegates of the Illinois State Medical Society, it was voted to continue the study of a plan, suitable for the state of Illinois and to report at the 1941 meeting of the Society.

The Committee on Medical Preparedness of the American Medical Association is sending to every physician in the United States in regard to his fitness, experience, availability and desire as to military service in the event of war. This is in answer to a request from the War Department of the United States, presented at the meeting of the 1940 House of Delegates of the AMA. Some of the delegates were openly curious as to the need of such a questionnaire at this time and wondered whether it was the result of the War Hysteria sweeping the country, particularly the East at that time or whether it was part of a great plan of war propaganda. However, since it was the request of the War Department that the Medical Profession cooperate in this manner, no objections were offered to the plan and a Committee under Past President Abell was formed to carry on the work. This questionnaire is the beginning of their work. We feel that every physician in the United States should fill in the same as carefully, accurately and completely as he possibly can. If he is undecided as to whether he wishes to volunteer for military service in the event of war, he can answer "undecided at this time." Thus he is no way obligated and is wholly cooperated with the desires of his Government.

E. S. Hamilton, M. D.,
Chairman.

Correspondence

MEDICAL PREPAREDNESS

To: Secretaries of County Societies
Illinois State Medical Society:

You have all received the letter and request for information sent out by the Committee on Medical Preparedness of the American Medical Association at the request of the House of Delegates as our part in a national survey to determine certain facts desired at this time. Several resolutions were introduced before the A.M.A. House of Delegates, (one by an officer-member from the United States Army) urging the medical societies of the country aid the Government in procuring certain information concerning every physician of the country so that in an emergency, the medical profession could be organized without loss of time.

The House of Delegates selected a Committee on Medical Preparedness with Dr. Irvin Abell of Louisville as Chairman, with a membership of ten from all parts of the country, in addition to five A.M.A. officials as ex-officio members. This Committee has already conferred with Governmental officials in Washington and other meetings will be held in the near future. In the meantime we have been urged to fill out the schedule submitted so that the information may be on file at A.M.A. headquarters and transferred to punch cards.

All information submitted by physicians on these schedules will be kept in strictest confidence, and it is important that every physician in Illinois fill out their schedule as soon as possible. We would respectfully urge that each of you refer to this survey at your next meeting or when conferring with members of your society, asking each physician to complete the schedule and return it to the A.M.A. Committee on Preparedness immediately.

You will no doubt remember that during the World War no less than 31,000 physicians of our country enlisted in the Army Medical Corps, while there were between two and three thousand physicians in other branches of the service. In the event of another war, we all know that the medical profession will do its duty, and once more there will be a satisfactory response on the part of the members of our profession.

Each week there is a special section in the

Journal of the A.M.A. relative to Medical Preparedness which we trust you will follow carefully during the coming months.

Trusting you will endeavor to get 100 per cent response on the part of your members in filling out their schedules, and with kindest regards, I am,

HAROLD M. CAMP, M. D.,
Secretary.

AMERICAN CONGRESS OF PHYSICAL THERAPY TO CONDUCT INSTRUCTION COURSE

The 19th annual scientific and clinical session of the American Congress of Physical Therapy will be held September 2 to 6th, inclusive, at Hotel Statler, Cleveland, Ohio. This year there will be a departure from the usual arrangements in that the mornings will be devoted to an instructional seminar with the scientific program presented afternoons and evenings. This enables physicians to economize on time by attending both the instruction course and the annual convention during the same week. The entire instruction schedule is elective in character. Registrants may pursue only the individual courses they desire. The complete course consists of twelve lectures from a diversified list of forty-eight. The scientific program itself consists of papers, demonstrations and motion pictures covering every branch of physical therapy. There will be separate scientific program covering Eye, Ear, Nose and Throat subjects. Write for schedule, fees, etc., to the American Congress of Physical Therapy, 30 North Michigan Avenue, Chicago, Ill.

EXTRA COPIES AVAILABLE OF THE CENTENNIAL NUMBER OF THE JOURNAL

We have on hand a goodly number of the Centennial issue (May, 1940) of the ILLINOIS MEDICAL JOURNAL. This particular number records the progress of medicine in Illinois during the past hundred years. It is really a granary of medical historical data. Postage, 10 cents. 6221 Kenmore Avenue, Chicago, Illinois.

HAVE YOU READ THEM?

Nation's Business, May, 1940, issue, contains an article entitled "The Case of Private MEDICINE." The editor of the publication, Merle Thorpe says: "If the people understand the danger facing private medicine, they can be counted upon to act promptly." Reprints of the "CASE OF PRIVATE MEDICINE" may be obtained at a cost of ten cents per copy by addressing NATION'S BUSINESS, Washington, D. C. Also in reprint form can be had from the same source a pamphlet captioned, "Health by POLITICAL DEGREE."

THE AMERICAN COLLEGE OF PHYSICIANS

The Twenty-Fifth Annual Session of the American College of Physicians will be held in Boston, with general headquarters at the Statler Hotel, April 21-25, 1941.

Dr. James D. Bruce of Ann Arbor, Mich. is President of the College and will have charge of the program of general scientific sessions. Dr. William B. Breed of Boston has been appointed General Chairman of the Session, and will be in charge of the program of clinics and demonstrations in the hospitals and medical schools and of the program of panel and round table discussions to be conducted at the headquarters.

ANNUAL MEETING OF EYE AND EAR SPECIALISTS

The American Academy of Ophthalmology and Otolaryngology will hold its forty-fifth annual convention in Cleveland, October 6 to 11, with headquarters at the Hotel Cleveland.

The Academy, an organization of more than 2,500 specialists in diseases of the eye, ear, nose and throat, carries on an active program of education for its members. In addition to scientific papers, an elaborate series of courses is presented at each convention to bring the members up to date in their chosen fields. More than 100 of these teaching lectures will be offered this year.

In the past year arrangements have been made to extend the teaching activities to young physicians just entering on specialization. Home study courses are being prepared for any of these young men who wish to take them and their work will be supervised by members of the academy interested in improving the caliber of specialists in practice.

The Cleveland meeting will be noteworthy in several respects.

The Academy will honor Dr. Secord H. Large, Cleveland, who this year completes thirty years as comptroller of the organization. Dr. Large as the honor guest of the meeting will receive many special distinctions.

Immediately following the Academy meeting, there will be a Pan-American Congress for Ophthalmology, October 11 and 12, which eye specialists from all the Latin American countries are expected to attend.

Dr. Frank Brawley, Chicago, is president of the Academy and Dr. Frank R. Spencer, Boulder, Colo., is president-elect. Vice presidents are Drs. Arthur W. Proetz, St. Louis; Joseph F. Duane, Peoria, Ill., and Charles T. Porter, Boston. Dr. William P. Wherry, 1500 Medical Arts Building, Omaha, is executive secretary.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The annual written examination and review of the case histories (Part I) for Group B candidates will be held in various cities of the United States and Canada, on Saturday, January 4, 1941, at 2:00 P. M. Candidates who successfully complete the Part I examinations proceed automatically to the Part II examinations held later in the year.

The following action regarding case records to be submitted by candidates taking the Group B, Part I, examination was passed by the Board at its annual meeting in Atlantic City, N. J., on June 6, 1940: "Case records submitted by candidates must be of patients treated within four years prior to the date of the candidate's application. The number of cases taken from one's residency service should not be more than half (25) of the total number of fifty (50) cases required."

Applications for admission to Group B, Part I, examinations must be on file in the Secretary's Office not later than October 5, 1940.

The general oral and pathological examinations (Part II) for all candidates (Groups A and B) will be conducted by the entire Board, meeting at Cleveland, Ohio, immediately prior to the June 1941 meeting of the American Medical Association.

After January 1, 1942, there will be only one classification of candidates, and all will be required to take the Part I and Part II examinations.

For further information and application blanks, address Dr. Paul Titus, Secretary, 1015 Highland Building, Pittsburgh (6), Pennsylvania.

Training Requirements

In response to numerous inquiries regarding special training requirements, the Board desires again to announce that there are three methods of meeting these requirements for admission to the Board examinations. First, by the residency system; second, by the partial residency and partial assistantship method; and third, entirely by the assistantship or "preceptorship method." Details of the residency requirements are given in the Board booklet. The Board will accept in lieu of the formal residency service the training acquired by a candidate serving on an assistant or dispensary staff of an obstetrical and gynecological division of a recognized Hospital, under the direction of a recognized obstetrician-gynecologist (preferably a Diplomate). The time required for this type of training must be longer than with the formal, more intensive residency type of training, and the allowance of time depends upon the duties and responsibility given the candidate. Applicants lacking all formal special training should have a minimum of five years of hospital clinic, or assistant staff appointments in the specialty, under approved direction. Teaching appointments without accompanying hospital staff or clinical appointments will not satisfy the Board requirements. A special form amplifying the original application must be filled out to cover the details of such assistantship, or preceptorship type, of training. The Board approves for special training, work done in institutions approved jointly by the Board and by the Council on Medical Education and Hospitals of the A. M. A.

THE ELIZABETH McCORMICK CHILD RESEARCH GRANT OF THE INSTITUTE OF MEDICINE OF CHICAGO

The Committee on The Elizabeth McCormick Child Research Grant of the Institute of Medicine of Chicago has at its disposal the sum of $1,500 which may be awarded to qualified investigators in the Chicago area for the aid of research in child welfare. Projects should in a broad sense be in the field of pediatrics. Applications will be received up to October 15, 1940, and the award made soon after. Address all communications to Dr. John Favill, Secretary of the Elizabeth McCormick Child Research Grant, 122 South Michigan Avenue, Chicago. Since there are no formal blanks, applications should be made by letter.

EXAMINATION BY AMERICAN BOARD OF OPHTHALMOLOGY

THERE WILL BE ONLY ONE WRITTEN EXAMINATION DURING 1941

This will be held in various cities throughout the country on March 8th.

Candidates enrolled in the Preparatory Group who have been advised that they will be eligible for examination during 1941 should make application AT ONCE to take this written examination.

Application must be made on the regular blanks provided for the purpose and must be received in the Board Office before DECEMBER 1st, 1940.

ORAL EXAMINATIONS 1941

Cleveland, May or June

October (place to be announced later)

DEADLINE FOR CASE REPORTS

February 1st

July 1st

A special oral and clinical examination will be held on the Pacific Coast during 1941 providing there will be enough candidates to warrant it. Applications for this examination should be filed before September 1st, 1940, so that the Board may complete necessary arrangements.

If you plan on taking your examination during 1941, please write at once to the Board Office for formal application blanks, indicating your preference of examination place.

AMERICAN BOARD OF OPHTHALMOLOGY

6830 Waterman Avenue,

St. Louis, Missouri.

WINNER OF THE 1940 MISSISSIPPI VALLEY MEDICAL SOCIETY CONTEST ANNOUNCED

The third annual Essay Contest of the Mississippi Valley Medical Society "for the best unpublished essay on a subject of practical and applicable value to the general practitioner of medicine" has been concluded. The Annual Awards Committee of the So-

ciety has announced that John F. Casey, M. D., of Boston, Mass., Visiting Physician, St. Elizabeth's Hospital, Boston, is the winner, F. Stanley Morest, M. D., of Kansas City, Mo., second, and Charles W. Pavey, M. D., Columbus, O., third. The winner receives $100.00 cash prize, a gold medal, a certificate of award, and an invitation to present his essay before the annual meeting of the Mississippi Valley Medical Society. Certificates of merit will be given to Doctors Morest and Pavey. Dr. Casey will address the Society on the subject of his winning essay, "A Study of the Use of Sulfapyridine and Sulfathiazole in Pneumonia with Particular Reference to the Treatment of Pneumonia by the General Practitioner," at Rock Island, Ill., Sept. 26. His paper, and probably those of Drs. F. S. Morest and Charles W. Pavey, will be published in the Jan. 1940 issue of the *Mississippi Valley Medical Journal*, the Society's official publication. The winner last year was Dr. Frederick F. Boyce, of New Orleans, Ass't. Prof. of Surgery, Louisiana State University Graduate School of Medicine, for his essay "Toxic Thyroid Disease as a Surgeon Would Have the General Practitioner Conceive It, with a Special Note on the Liver Factor," which appeared in the Jan. 1940 issue of the Mississippi Valley Medical Journal. The M.V.M.S., Essay Contest has now been established as an Annual Affair, but the rules for the 1941 contest will not be available until December 1.

SIXTH ANNUAL MEETING, MISSISSIPPI VALLEY MEDICAL SOCIETY, ROCK ISLAND, ILL., SEPTEMBER 25-27

The Sixth Annual Meeting of the Mississippi Valley Medical Society, "The Mid-West's Greatest Intensive Post-Graduate Assembly for General Practitioners," will be held at the Hotel Fort Armstrong, Rock Island, Ill., Sept. 25-26-27. The program will be given by 32 clinician-teachers who will give over 60 lectures, demonstrations, round table discussions, etc. A special feature of the program this year will be numerous short instructional courses, there being 41 of these scheduled. On September 25 there will be an All-Chicago program conducted by 10 well known clinician-teachers from the medical schools there, concluding with a complimentary Stag Supper. On Sept. 26 the annual banquet will be held with Dr. N. B. Van Etten, of New York City, President of the American Medical Ass'n., and the Presidents of Illinois, Missouri, and Iowa State Medical Societies as speakers. On Sept. 27 the short instructional courses will be given by faculty members from the University of Iowa. There will be a big technical and scientific exhibit hall. Every ethical physician is cordially invited to attend. The complete program appears in the September issue of the *Mississippi Valley Medical Journal* and further information may be secured from the Secretary, Harold Swanberg, M. D., W. C. U. Building, Quincy, Illinois.

POSTGRADUATE COURSE WELL ATTENDED

The following doctors attended the special 1940 summer postgraduate courses offered in Obstetrics and Pediatrics at the University of Illinois College of Medicine:

D. J. Freriks, La Grange.
W. W. Keene, Yates City.
J. C. McMillan, New Berlin.
W. I. Lewis, Herrin.
W. B. Fonvielle, Rockford.
R. E. Hunt, Springfield.
C. K. Carey, Rushville.
P. A. Gannon, Pontiac.
H. C. Sauer, Fairbury.
J. E. Walton, Homer.
R. C. Heiligenstein, Belleville.
J. W. Daugherty, Chillicothe.
E. S. Shonyo, Elgin.
E. E. Davis, Avon.
E. T. Lark, Columbia
Nicholas Lucia, Braidwood.
R. C. Aikin, Blue Island.
J. J. Klein, Harrisburg.
J. H. Fowler, Moline.
R. F. Snider, St. Francisville.
E. W. Burroughs, Ridgway.
W. J. Houghton, Riverside.
W. H. Schuette, Mason City.
Paul Hochberg, Seaton.
R. D. Martin, Sullivan.
E. A. Wester, Mt. Sterling.
Myron Boylson, Tuscola.

DOCTOR'S WIFE HAS HER OWN TEN COMMANDMENTS

She must not know the meaning of the word "jealous."

She must never gossip.

She must run a cafeteria, serving meals at all hours for her husband.

She must be—like Caesar's wife—above reproach.

She must have self-reliance and self-control.

She must be able to think quickly and sanely in emergencies.

She must be a diplomat, see all, hear all, say a lot, yet say nothing.

She must learn to bear stoically and without complaint, disappointments in her personal plans.

She must be a good mother and father, because doctors are often too busy to discipline their own children.

She must be a good "doctor" because doctors never take time to doctor themselves.

—Author Unknown,
Wichita Medical Bulletin.

The Salvation Army has announced that it was dispensing with its few remaining horses and wagons in eleven eastern states. The announcement ended with a note of suppressed indignation: "We have raised a generation of men who can't drive, feed, or care for a horse."—*The National Humane Review.*

Original Articles

THE ROLE OF THE KIDNEY IN CARDIO-VASCULAR-RENAL DISEASE

L. G. ROWNTREE*, M. D.

Philadelphia Institute for Medical Research
Philadelphia General Hospital

PHILADELPHIA, PENNSYLVANIA

Mr. President, Fellow Practitioners, Ladies and Gentlemen:

It is a distinctive honor indeed to be invited to deliver the oration in medicine at the Hundredth Anniversary of the Medical Society of the State of Illinois. I wish to thank your President and Program Committee for considering me worthy of participating in your proceedings on so auspicious an occasion.

Before proceeding to our main discussion I believe that it would be appropriate and perhaps entertaining and instructive to turn back a century in time in order to take a glance at organized medicine as it existed in Illinois one hundred years ago, and also to catch a glimpse of medicine and physiology as they concerned the kidney around the year 1840.

Your President informs me that the Illinois State Medical Society was formally organized in the year 1840 and that the presiding officers continued in office until the year 1850. Concerning their activities during this decade I unfortunately could find no report. The first reference that I can find, to the proceedings of your organization pertains to a meeting held in Springfield, Illinois on June 4th, 1850. On a motion by Dr. William Herrick of Chicago—Dr. Rouse of Peoria was voted into the chair. He in turn appointed a nominating committee and as a result Dr. Herrick was elected President, and Dr. Meek of Chicago Secretary.

The Transactions of 1852 indicate that the meeting was held in Jacksonville and that Dr. Samuel Thompson presided. Dr. N. S. Davis was appointed to deliver the Oration in Medicine at the next annual meeting. Your society got right down to real business. It made an assessment of $2.00 a year against each of its 80-90 members. It also called on the carpet and censured an oculist, one Dr. J. W. Halsted, for advertising in a local paper. Having disposed of these important matters the society was ready to proceed with the scientific session. The first paper by Dr. Cooper of Peoria dealt with "Collodion as a remedy in entropion." Another paper by Dr. Hall considered "The use of citric acid in rheumatism."

In 1853 the meeting was held in Chicago and Dr. N. S. Davis gave a broad, comprehensive and scholarly address on "The intimate relation of medical science to the whole field of Science." He presided at the annual meeting held in Vandalia in 1856. Since no greater organizer is to appear in your society nor in the ranks of American Medicine in general, we will leave you in the able hands of that great man, Dr. N. S. Davis, who is usually regarded as the father of the American Medical Association.

Now let us consider medicine of a hundred years ago as it pertained to diseases of the kidney. Richard Bright, the greatest student of nephritis that the world has ever known, was still carrying on his investigations on kidney diseases in London in the wards of Guy's Hospital, in the laboratory, and the autopsy room. His laboratory equipment consisted of a spoon and a candle. (Fig. 1.) With this simple equipment he established once and for all, the relationship of albuminuria to diseases of the kidney. In his laboratory was also studied the role of urea in nephritis by one of his associates. Owen Rees. Bright had uncanny insight and seems to have visualized in his day all of the important problems of the kidney, and anticipated all of the important lines which investigations have taken in the attempt to solve the many problems involved in cardiorenal-vascular disease. Bright had the happy faculty in investigation of setting his own feet in the right path, thereby leading his followers into fruitful avenues of research.

But despite Bright's epoch-making discoveries the profession was slow in recognizing their great significance. Many American Text-books of medicine of a century ago entirely overlooked Bright and his ideas of nephritis. Thus Mackintoshes "Practice of Physic" 1837 and Hosack's "Lectures on the Theory and Practice of Physic," 2nd American Edition, 1838, carry no mention of Bright or his work and evidence no insight whatsoever into the problems of nephritis. However, in Bigelow and Holmes' American edition of Marshall Hall's "Practice of

Oration in Medicine, I. S. M. S., May 22, 1940.

*The author wishes to express his gratitude to the American Philosophical Society for financial support from the Daland Fund.

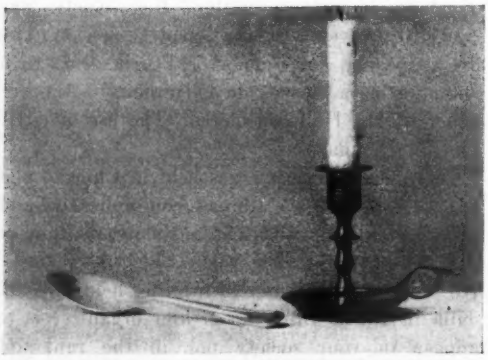


Figure 1. The type of laboratory equipment used by Bright.

Medicine" 1839, they refer to Bright's contributions and quote him extensively. They state that Bright was the first to describe the granular kidney, though he gave it no name, that prior to Bright such a kidney was regarded as a subsidiary of dropsy, whereas now "dropsy is no more than one of its symptoms." They further state, with rare insight, that it "tends to impoverish the blood by depriving it of its coloring matter, i.e., production of anemia."

Physiology at this time was in even a more deplorable state. Though Bowman was at work, he had not yet published his studies on the kidney, so nothing of any consequence was known as to the secretion of the urine or the physiology of the kidney.

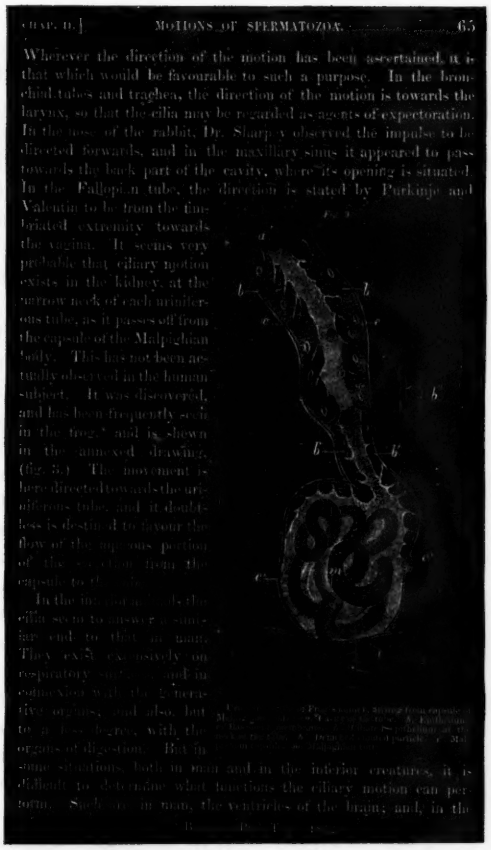
It is interesting to know that Hippocrates was altogether ignorant of the fact that the urine was excreted by the kidney. Galen recognized the truth and in his book on "The Natural Faculties" taught that the urine was separated from the blood by the kidneys and carried to the bladder by the ureters. Though he described his own experiments which proved this contention beyond a question of doubt, he complained most bitterly that the medical profession of his day would not accept his teachings and in fact were not sufficiently interested to even observe his experimental evidence.

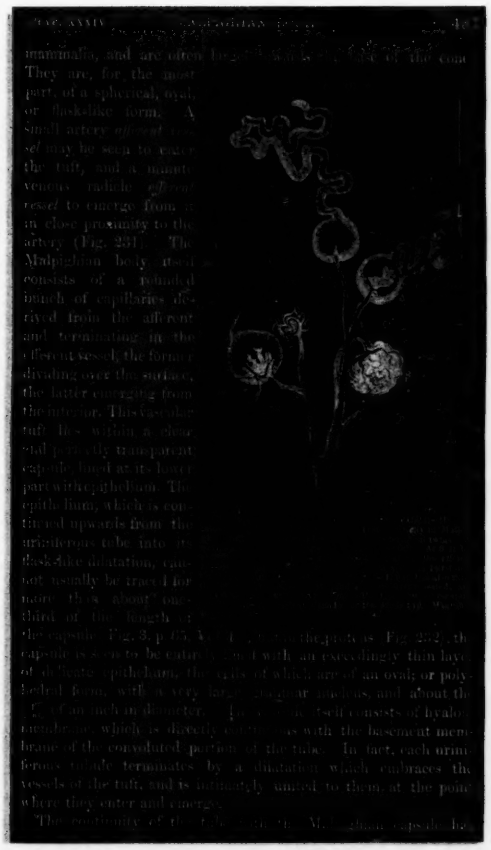
Two works on "Human Physiology" appeared in 1840. Elliotson disposed of urinary secretion by saying that it is purely excrementitious but he does give an interesting list of urinary constituents. Dunglinson says "with regard to the mode in which the operation is affected we are in the same darkness that hangs over the glandular secretions in general."

The brilliant illumination that has flooded renal physiology during the last century was touched off by the torch in the hands of William Bowman. (Fig. 2). Working in London he investigated the structure of the kidney. In studying the tubules he noted that the basement membrane was continued on, and enveloped the glomerular tuft. (Fig. 3). Later in injection experiments he noted that the Malphighian body did not always fill the capsule and that substances, dyes—injected into the renal artery—passed into the glomerulas vessels and appeared in the efferent veins, but also that dyes passed over into the interior of the capsule. (Fig. 4 and 5). And then came his epoch-making speculations which started the world on the study of urinary secretions.

"Reflecting on this remarkable structure of the Malpighian bodies, and on their singular connections with the tubules, I was led to speculate on their use. It occurred to me that as the tubes and their plexus of capillaries were probably, for reasons presently to be stated, the parts concerned in the secretion of that portion of the urine to which its characteristic properties are due (the urea, lithic acid, etc.) the Malphighian

Dr. Wm. Bowman





Bowman's Experiments 3 and 4

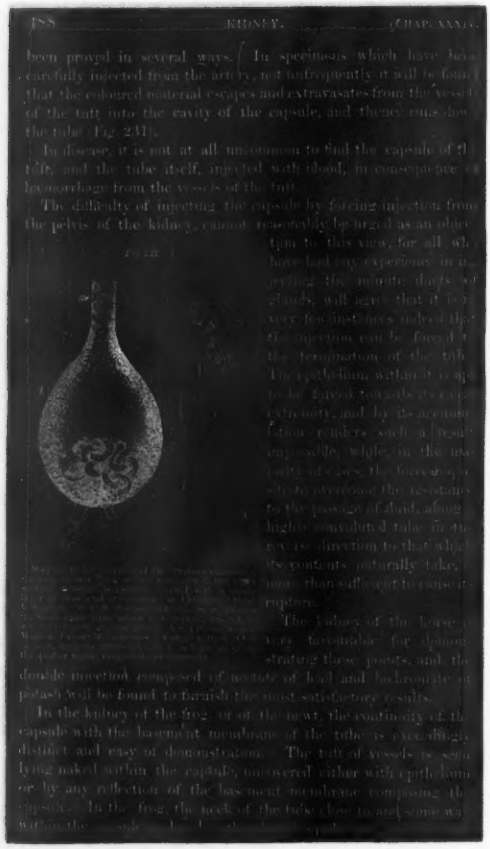
bodies might be an apparatus designed to separate from the blood the watery portion."

Here for the first time we see an attempt to explain the urinary secretion on a rational basis. Also we see in his picture of renal units so many of the elements concerned in nephritis and hypertension—glomeruli, tubules, arterioles and capillaries. Modern medicine attempts to apply current concepts of the function of these structures to renal physiology and pathology.

The function of the kidney in health and disease.

In order to understand the function of the kidney in disease we must for a moment get a glimpse of its function in health. The function of the kidney is to help maintain homeostasis in the body through the elimination from the blood to the urine of excessive superfluous and waste products. These functions embrace (1) the excretion of water, salt, and the maintenance of water balance of the body. (2) The excretion of end products of metabolism. (3) The maintenance of acid base equilibrium. (4) Synthesis, as exemplified in hippuric acid, and (5) the control of hormonal and vitamine content of the blood.

By the aid of a simple scheme (Fig. 6) prepared some years ago by Dr. Philip Hench of the Mayo Clinic, it is possible to grasp at a glance the development of the different theories of renal physiology of the last century in relation to the mechanism of excretion of urine. Bowman in 1842 stated that urine was excreted by the simple process of secretion on the part of both the glomeruli and tubules, solids by the tubules, water by the glomeruli. Ludwig favored mechanical filtration through the glomerulus and reabsorption by diffusion in the tubules. Heidenhain thought it all a matter of the vital process of secretion. Cushny introduced the



Bowman's Experiment 5

concept of the mechanical filtration through the glomerulus of deproteinized plasma and the vital reabsorption of Locke's solution by the tubules. Starling and Verney accepted this viewpoint but maintained that the tubules were capable of secreting additional urea and certain dyes into the urine. The work of E. K. Marshall on the aglomerular fish would seem to prove this contention, at least so far as certain dyes are concerned. This point of view is accepted by A. N. Richards. It would seem, therefore, that the mechanism of urinary excretion is at last fairly well established.

Based on the modern concept of urinary secretion, a really tremendous load of work is thrown upon the kidney, as indicated by the accompanying table from Cushny. (Table 7). The purpose of renal activity is not just the excretion of urine but rather the clearing and purifying of the blood. It is estimated that 90 litres of plasma are cleared and purified in the production of one litre of urine—eighty-three litres is water and all but one litre of this is reabsorbed in the tubules together with glucose, salt, urea, uric acid, potassium, phosphates, sulphates, etc.

Since the kidneys function by night as well as by day their work is not only enormous but also continuous. While it is true that certain glomeruli and their accompanying tubules may at times be at rest temporarily, the kidney as a whole—like Tennyson's brook—goes on forever. The continuity of action constitutes a constant and considerable strain and this may be a factor in bringing on nervous and vascular reactions within the kidney and extra-renally which may play a role in cardiorenal-vascular disease. Since renal ischaemia has been found by Goldblatt to result in hypertension, the question arises with me as to whether a fatigued kidney may close off an excessive number of glomeruli and tubules, thereby automatically inducing renal ischaemia, which in turn leads to increase in blood pressure. This, of course, is merely an untried suggestion.

Renal Functional Tests.—For many decades after Bright, medicine centered its interest on the clinical manifestations of kidney disease, on urinalysis, and on necropsy findings, both gross and microscopic. A recent study by Christiernin, Dublin and Marks of the Metropolitan Insurance Company indicates that in a large way the seriousness of the presence of albumin and casts in the urine is well understood. Thus in standard risks, in predicting the outcome in patients showing albuminuria or casts they have

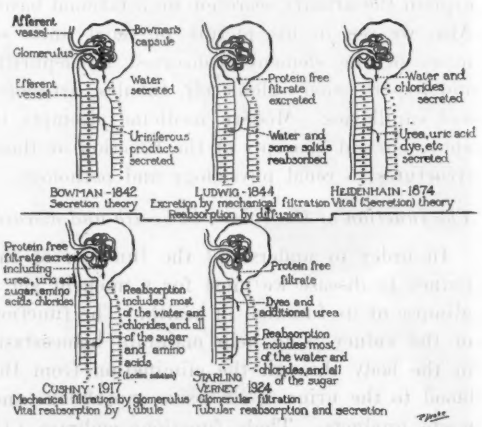

Afferent vessel
Glomerulus
Efferent vessel
Bowman's capsule
Water secreted
Urinferous products secreted
BOWMAN - 1842
Secretion theory
Protein free filtrate excreted
Water and some solids reabsorbed
LUDWIG - 1844
Excretion by mechanical filtration
Reabsorption by diffusion
Water and chlorides secreted
Urea, uric acid dye, etc secreted
HEIDENHAIN - 1874
Vital (Secretion) theory
Protein free filtrate excreted including urea, uric acid sugar, amino acids chlorides
Reabsorption includes most of the water and chlorides, and all of the sugar and amino acids
(Locke solution)
CUSHNY - 1917
Mechanical filtration by glomerulus
Vital reabsorption by tubule
Protein free filtrate
Dyes and additional urea
Reabsorption includes most of the water and chlorides, and all of the sugar
STARLING VERNEY - 1924
Glomerular filtration
Tubular reabsorption and secretion

Figure 6. A schematic presentation of the physiology of urine secretion as prepared by Dr. Philip Hench.

COMPOSITION OF GLOMERULAR FILTRATES AND REABSORBED FLUID

	90 Litres Plasma contains		83 Litres Filtrate	82 Litres Reabsorbed Fluid contain		1 Litre Urine contains	
	Per cent.	Total	contain	Per cent.	Total	Per cent.	Total
Water	92	83 litres	83 litres	.....	82 litres	95	950 c.c.
Colloids	7.5	6750 gr.	.....	.....	.....	....	.....
Glucose	0.1	90 gr.	90 gr.	0.11	90 gr.	....	.....
Sodium	0.3	270 gr.	270 gr.	0.32	266.5 gr.	0.35	3.5 gr.
Chloride	0.37	333 gr.	333 gr.	0.4	327 gr.	0.6	6.0 gr.
Urea	0.03	27 gr.	27 gr.	0.008	7 gr.	2.0	20.0 gr.
Uric Acid	0.004	3.6 gr.	3.6 gr.	0.003	3.1 gr.	0.05	0.5 gr.
Potassium	0.02	18.0 gr.	18.0 gr.	0.02	16.5 gr.	0.15	1.5 gr.
Phosphate	0.009	8.1 gr.	8.1 gr.	0.0008	6.6 gr.	0.15	1.5 gr.
Sulphate	0.002	1.8 gr.	1.8 gr.	.....	.....	0.18	1.8 gr.

been eminently satisfied but in substandard risks showing albuminuria, in cases showing both albuminuria and casts, and especially in those cases where overweight or hypertension are also present, prediction is unsatisfactory.

TESTS OF RENAL FUNCTIONS

A. Tests of Excretory Capacity

1. Water (Albarron).
2. Chemicals—potassium iodide, lactose, salicylates, sodium chloride urea-sugar following phloridzin.
3. Enzyme—diastase (Wohlegemuth).
4. Dyes—methylene blue, indigo carmine, rosanaline Phenolsulphonphthalein (1910).

B. Tests of Concentration

Concentration in the blood of:

1. Ions—by electrical conductivity.
2. Molecules and ions—by cryoscopy.
3. Cholesterol.
4. Total incoagulable nitrogen—total non-protein nitrogen.
5. Urea—Doremus, Marshall, Hench, Van Slyke and Austin, Carr.
6. Creatinine, uric acid, phosphorus, sulphates.

C. Rates of coefficients of secretion of:

1. Urea—Ambard, Van Slyke.
   Urea clearance test.
2. Phenolsulphonephthalein—Shaw, Habein, Rowntree, Chapman and Halsted.
3. Creatinine filtration or clearance—Rehberg.
4. Sulphates—Wakefield, Keith and Macy.

D. Miscellaneous Tests

1. Urinary concentration relative to food and water intake—Schlayer and Hedinger—Mosenthal, Newburgh.
2. Urea ratio—Mosenthal and Bruger.
3. Synthesis as exemplified in Hippuric acid—Kingsbury.

Towards the end of the last century and in the beginning of this one, the idea developed that it should be possible to test the function of the kidney quantitatively in health and disease. Hence tests of renal function were introduced. Originally these tests concerned the excreting capacity of the kidney (Table 8a) and dealt exclusively with the urine. Later they dealt with the mounting concentrations or accumulations of substances (Table 8b) in the blood which because of diminished renal function had failed to be excreted, and finally tests were employed to reveal the coefficients or rates of excretion (Table 8c). During the last thirty years renal functional tests have come to occupy a place of paramount importance in the investigations of disease of the kidney.

The tests of preference at the present time are, the urea clearance test of Van Slyke and the dye tests (phthalein test or PSP) of Rowntree and Geraghty as modified by Shaw; the creatinin clearance test of Rehberg (indicating to a large extent glomerular activity) and tests involving the quantity and specific gravity of urine as devised by Mosenthal and by Newburgh. These procedures give an uncanny insight into the functional efficiency of the kidney. Thirty years have elapsed since we introduced the phenolsulphonphthalein test which constituted the first test of renal function of true practical clinical value.

In recent years it has become apparent that a judicious combination of tests yields the most accurate index to renal function. Thus in the March number of the Annals of Medicine, McGee and Martin, who were attempting to find the best combination of tests concluded that:

(1) No one test is adequate.

(2) The fifteen minute excretion of PSP (after intravenous) injection yields the expected results most often.

(3) PSP can not be displaced by any available procedure in convenience or accuracy.

(4) When combined with urea clearance test —confirmation of renal disease was found in all but 6 of 290 patients.

Classification of Nephritides.

As time has passed numerous pathologists, clinicians and investigators have felt a growing need for further classifying the various forms of nephritis and diseases of the kidney. Some classifications have been based solely on clinical grounds, some on pathologic findings, others on

CLASSIFICATION OF CHRONIC NEPHRITIS

Senator	Aschoff	Addis
1. Parenchymatous without induration	Nephropathia degenerativa	Degenerative Bright's Disease (Nephrosis lipoid or emyloid)
2. Interstitial with induration	Nephropathia inflammatoria	Hemorrhagic nephritis (Glomerulonephritis)
a Primary		
b Secondary		
c Arterioscerosis		
3. Diffuse combination form	Nephropathia circulatoria	Arteriosclerotis Bright's Disease (Nephrosclerosis)
Inflammatory—(Glomerular destruction) Degenerative—(mostly tubular)	Circulatory—(thickening of small renal arteries)	

Volhard and Fahr (Clinical and Pathologic)	Christian (Clinical)	Widal and Javal (Functional)
Acute	Acute	
Acute		
a) Diffuse glomerular		
b) Focal glomerular		
c) Nephrosis (tubular)		
Chronic nephritis	Chronic	
a) Chronic diffuse glomerular nephritis, early chronic or late terminal	a) with vascular hypertension	a) Azotemic
b) Chronic nephrosis (tubular)	b) with edema	b) Chloremic
c) Mixed a and b	c) Mixed or combined type	c) Mixed
d) Chronic focal	d) Renal arteriosclerosis progressing to chronic nephritis	
e) Arteriosclerotic nephritis Nephritis secondary to vascular change	e) Essential vascular hypertension progressing to chronic nephritis	
f) Chronic diffuse glomerular secondary to malignant sclerosis, giving same renal picture as "a"		

functional derangement as evidenced by chemical studies. The outstanding classifications at present are those suggested by Senator, Aschoff, Addis, Volhard and Fahr, Christian and by Widal and Javal. (Table 9).

Volhard and Fahr emphasized the necessity of differentiating from glomerulonephritis the pathologic processes found in the tubules and those encountered in the vascular diseases as exemplified in benign and malignant hypertension. The latter was made possible in part by the employment of renal functional tests, the sphygmomanometer, and the ophthalmoscope. Their classification unquestionably constitutes a milestone in the development of our knowledge of nephritis, as it led to more intensive investigations into all its various forms. However, as a classification it is not altogether perfect. The criticism of Aschoff—the leading pathologist of his day, is subtly revealed in his use of the term "nephropathia" instead of nephritis, and of Addis, America's most careful student of nephritis, by his adherence to the old term "Bright's disease" instead of nephritis for forms other than the glomerular or hemorrhagic form. All three, Volhard and Fahr, Aschoff, and Addis, however, agree on the desirability of differentiating degenerative and vascular forms from inflammatory glomerular nephritis. Christian, who may justly be regarded as one of our soundest students of the subject, in both clinical and pathological fields, questions the existence of lipoid nephrosis as a separate disease representing tubular degeneration since he, like Bell, feels that many cases of this so-called lipoid nephrosis represent true glomeruler nephritis in a different phase. In fact Bell actually classifies lipoid nephrosis as a subdivision or form of glomerular nephritis. Christian also believes that we should not forsake the old term "Bright's Disease." Widal and Javal recognize two varieties of nephritis, an azotemic and chloremic form. All of these authorities concur in the belief that mixed or combined forms of nephritis are of common occurrence.

The correlation of some of these forms of nephritis is ingeniously revealed in the arrangement presented by Weiss (Table 10.) It is a matter of general agreement that in inflammatory nephritis, glomerular destruction predominates, that in true degenerative nephropathies, the tubular changes are paramount, and that in circulatory or vascular forms of Bright's disease, the renal arteries, large and small, and the arterioles (and perhaps the afferent vessels to the glomeruli) are principally concerned, that the disease is widespread and general throughout

the organisms leading to death as a rule by injury other than that directly to the kidney.

The Etiology and Pathogenesis of Nephritis. Obviously the cause of nephritis interests us most of all, but unfortunately neither its etiology nor pathogenesis is clearly established to date. However, it has long been recognized that glomerular nephritis develops commonly as a sequela of certain acute infections involving the throat and upper respiratory tract. Unquestionably the haemolytic streptococcus is concerned etiologically in a large proportion of cases with the diffuse disease, as proven by recovery of the organism in culture, or its recognition through specific agglutinations and antistreptolysins, and also by the successful reproduction of the disease with haemolytic streptococcus in certain animals. Likewise the streptococcus viridans in subacute bacterial endocarditis is commonly associated with focal embolic glomerular nephritis. While much is known relative to the association of amyloid disease with chronic suppuration, little or nothing is known relative to the cause of lipoid nephrosis. Likewise the pathogenesis of arteriosclerosis, hypertension and cardiovascular-renal disease is still obscure.

Acute or Chronic Glomerular Nephritis. The acute form as a diffuse process, is most frequently seen in children as the sequela of scarlet fever, a septic sore throat, tonsilitis, or some other form of upper respiratory infection, due as a rule to streptococcus viridans and occurs as a complication of subacute bacterial endocarditis.

The onset of the diffuse form may be acute or insidious, the course mild or severe. Oliguria, haematuria, and oedema are almost constant as is also albuminuria. Hypertension likewise is extremely common. Renal insufficiency develops with great frequency, the level of the blood urea varying inversely with the quantity of urine excreted. Uraemia develops if the insufficiency is marked, and is characterized by stupor, twitching of the muscles, and often coma and convulsions. Despite the gravity of the clinical findings, recovery is the rule and a fatal outcome is the exception, the immediate mortality usually averaging less than 5 per cent.

The chronic form may follow an insidious onset or follow the acute onset and stormy course of acute nephritis. Latent periods or remissions are the rule, the disease progressing by successive acute exacerbations. Microscopic haematuria is the most constant finding, together with albuminuria and casts. Hypertension appears early as a rule and tends to be progressive. The disease tends to go in one of two directions—the hydropic (nephropathic) form in which oedema is marked or the azotemic (dry) form leading to uraemia. In many instances both tendencies are apparent. Acidosis also is very common, especially in the terminal stages.

In chronic glomerulonephritis the prognosis is bad. Once renal insufficiency is definitely established the end is frequently only a matter of months or a year or two at best.

The most important contribution to our

THE NOMENCLATURE OF CHRONIC KIDNEY DISEASE; THEIR RELATION TO ONE ANOTHER AND TO THE MAIN CLINICAL SYNDROMES OF BRIGHT'S DISEASE

Pathologic Era Wilks-Bartels (English) 1875-1900	Functional Era Widal (French) 1900-1925	Pathogenetic Era Volhard & Fahr (German) 1925	Clinical Classification Christian (American) 1925	Main Clinical Syndrome
Chronic parenchymatous nephritis	Chloremic (hydremic or hydropigenous)—(salt and water retaining)	Glomerulo-nephritis (nephrotic syndrome)	Chronic nephritis mith edema	Marked edema and pronounced albuminuria
		Chronic nephrosis		Absent (or slight) cardiovascular phenomena (no hypertension) cardiac hypertrophy, peripheral or retinal arteriosclerosis)
Chronic interstitial nephritis	Azotemic (nitrogen retaining)	Chronic glomerulonephritis	Chronic nephritis with edema	No nitrogen retention
		Nephrosclerosis or renal arteriosclerosis	Essential hypertension progressing into chronic nephritis	Recovery or death from intercurrent infection
				Hypertension and other cardiovascular phomena prominent
			Renal arteriosclerosis progressing into chronic nephritis	Nitrogen retention
				Albuminuria variable and edema not important
				Death from cardiac failure, cerebral vascular accident or renal failure (uremia)

Practical talks on Kidney Disease, Edmund Weiss. Charles T. Thomas, Publisher.

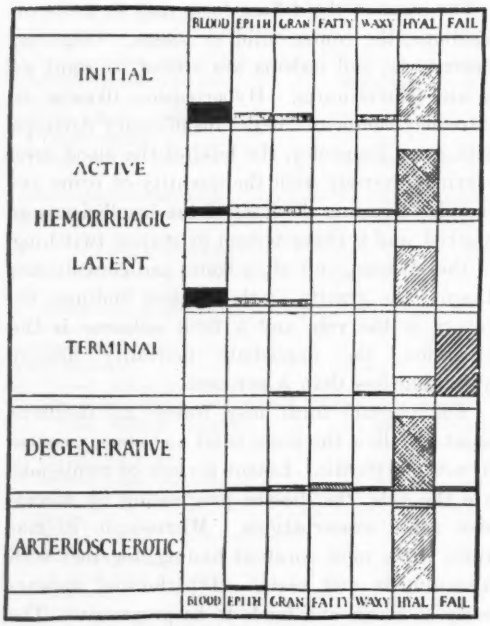


Chart 1.—Differential cast count.

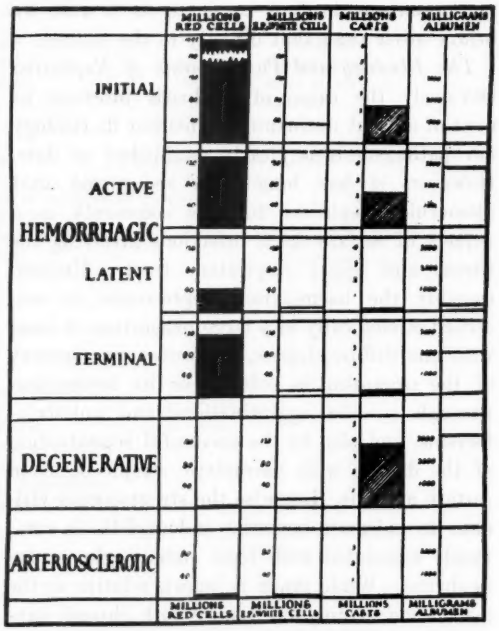


Chart 2.—Sediment and albumin (twelve hour urine).

Figure 11. From Addis's quantitative studies of the formed elements of the urine.

knowledge of this disease within recent years came from the work of Addis. He has studied the sediment of 12 hour urine collections microscopically and has quantitatively determined the number of red and white blood cells, epithelial cells and casts. The latter he has counted differentially and has emphasized the importance to prognosis of the presence of a large number of "Failure casts" which appear in the urine in the terminal stage of the disease. (Fig. 11.)

The fact that glomerular nephritis is so often the sequela of infection with streptococcus haemolyticus would suggest the possibility of its improvement through immunological measures and the prophylactic use of modern chemotherapeutic agents such as sulfanilimide and related compounds.

The Nephroses. Tremendous interest has centered on the lipoid nephroses during the last two decades, especially as the result of the studies of Epstein which indicated the diminished protein content of the plasma and the value of high protein feeding in its treatment. The cause is unknown. It is characterized by an insidious onset, generalized oedema and a waxy pallor. Albuminuria is marked, often extreme. The blood pressure remains normal. The blood chemistry is of great diagnostic significance but it is not specific. It consists of low serum proteins, especially of the serum albumin, and at times reversal in the albumin-globulin ratio. Lipaemia is present as is also a high level of cholesterol. The sedimentation rate is increased. Renal function usually remains normal, as indicated by urea clearance studies and dye tests. The prognosis is rather grave, though more than 50 per cent. recover. The high protein diet, the judicious use of diuretics and the removal of foci of infection have all proved of benefit in its management.

The plasma proteins have come into great prominence. Normally they total 6.3 to 8 per cent. Serum albumin usually averages 4 to 5 per cent. and the globulin 2 to 3 per cent. They yield an osmotic pressure of roughly 30 mm. Hg. Serum albumin exerts four times the osmotic pressure of the globulin. It fluctuates markedly and can be regenerated to the extent of 25 grams daily. As it decreases serum globulin tends to increase. Oedema results from bodily depletion of serum protein. At the critical point the total proteins are usually decreased to 5.5 per cent.,

FIGURE 12

CIRCULATORY NEPHROPATHIES

Senile Arteriosclerosis—chronic disease of the vessels of the aged with especial emphasis on vessels of kidney leading to renal insufficiency—uremia. Cause unknown.

Arteriolar Nephrosclerosis—10% leading to rental insufficiency. Cause unknown.

Benign Hypertension—hypertension may persist for many years—latent—or affecting heart or brain. Late albuminuria and cylindruria. Later symptoms and signs as in chronic glomerulonephritis and tendency to uremia.

Malignant Hypertension—more rapid course with more severe clinical manifestations and usually death within 2 years.

the serum albumin to 2.5 per cent. In the urine the albumin loss may amount to as much as 40 to 50 grams. Increase over normal in the urine usually indicates glomerular injury while the loss of more than one gram daily suggests degenerative disease.

Lipoid nephrosis is regarded by many as a degenerative process involving chiefly the tubules, by others as a general disease rather than a disease of the kidneys, by others, including Bell and Christian, as a transitional phase of glomerulonephritis.

Other clinical varieties of nephrosis exist, notably amyloid disease, which as a rule is secondary to poisoning with heavy metals, especially mercury. Experimental nephrosis is readily produced in animals with uranium, mercury and racemic tartrates. These forms of nephroses, however, do not represent lipoid nephritis. Similarly acute or chronic deficiency, in serum albumin can be induced by plasmaphaeresis but the resulting condition fails to constitute the lipoid form as encountered in the human.

Cardiovascular-Renal Disease. In this category we find the old time arteriosclerosis, arteriolar nephrosclerosis, essential and malignant hypertension. Irrespective of which is present the cause is unknown. (Fig. 12.)

Interest in newer phases of the subject should not lead us to lose sight of the importance of the old time arteriosclerotic kidney. An instance of this was recently encountered by the author in a case in which the attending physician stated that he had no idea just what was wrong with the old gentleman. It was a patient with an insidious onset of nausea and vomiting, a senile old man in whom a blood urea was well over 200 mg. per 100 cc. of blood, the condition rapidly ending in death.

Blood pressure studies and renal functional tests have aided in the differentiation of essential hypertension from true glomerular nephritis. Essential hypertension may persist for years doing but little harm, but later cripples the patient through the injury to eyes, heart, brain, or kidneys. The fulminating form of hypertension is truly malignant, killing surely and quickly, often in months, though occasionally patients survive as long as four or five years.

Janeway, one of our earliest and most outstanding students of cardiovascular-renal disease was among the first to recognize the widespread nature of the vascular changes and the frequency with which involvement of organs other than the kidney was abnormal at post mortem. The accompanying table (Fig. 13) appears in one of his articles published more than twenty years ago. Perhaps not more than 10 per cent. of such cases of cardiovascular-renal disease die of renal insufficiency.

A most intensive study of malignant hypertension has been carried on for many years at the Mayo Clinic by Keith, Wagner and Kernohan in which they have correlated clinical manifestations with the laboratory and autopsy findings. One of their slides (Fig. 14) in itself constitutes a whole lecture on the subject of diffuse arterial disease with hypertension. It ties up the progressive nature of the clinical manifestations with the progressive changes in the vessels, the fundamental process being the thickening of the arterial wall at the expense of its lumen, with consequent ischaemia leading to increase in blood pressure and eventually to fuctional and circulatory failure. In the study of this disease important information is derived

FIGURE 13

	Male Per cent. of total known—137		Female Per cent. of total known—47		
	No.	Per cent.	No.	Per cent.	Total
Gradual cardiac insufficiency	48	35.0	12	25.5	60
Uremic convulsions, coma, or gradual uremia	31	22.6	15	31.9	46
Cerebral apoplexy or its results	20	14.6	9	19.1	29
Angina pectoris	10	7.3	0	...	10
Edema of lungs	6	4.4	1	2.1	7
Progressive anemia	1	0.7	2	4.3	3
Pericarditis	1	0.7	0	...	..
Complicating acute infectious disease	9	6.6	4	8.5	13
Other accidental causes	7	5.1	2	4.3	9
Unknown	25	...	3	...	..
Sudden	4	2.9	2	4.3	6
	162		50		212

CLINICAL AND LABORATORY FINDINGS
DIFFUSE ARTERIAL DISEASE WITH HYPERTENSION
GROUP I (Ages 30-65)
GROUP II (Ages 21-55)
GROUP III (Ages 22-57)
GROUP IV (Ages 8-58)
VASOSPASTIC TYPE (Ages 24-39)
Chronic Glomerulonephritis (Ages 18-46)
SYMPTOMS
Hypertension often first observed during examination for early morning headaches, vertigo.
Hypertension often first observed during examination for early morning headaches, vertigo.
Usually increased nervousness, irritability, precordial distress, tachycardia, nocturia and dyspnea on exertion.
Nervousness, asthenia, loss of weight, palpitation, headaches, and dyspnea on exertion. Nocturia, and symptoms referable to various lesions of central nervous system.
Nervousness, asthenia, visual disturbances, headaches, diffuse pain in muscles.
Asthenia, anemia, headache and dyspnea on exertion. Nocturia.
RETINAL CHANGES
Usually limited to mild sclerosis of arterioles; minute exudates may be present.
Usually limited to moderate sclerosis of arterioles; thrombosis may be present.
Recurrent angiospastic retinitis with definite sclerosis in the arterioles, but without edema of disc.
Retinitis similar to that in renal arteriolar sclerosis, edema of the optic disc and surrounding retina. Arterioles markedly narrowed.
Retinal arteriolar morbidity with edema of disc and hemorrhagic retinitis. Tendency to arteriolar thrombosis.
Moderate vasospastic narrowing of arterioles in retina; infrequently terminal retinitis.
BLOOD PRESSURE (MOSTLY)
SYSTOLIC
DIASTOLIC
CARDIAC FUNCTION
Satisfactory
Often early impairment.
Often impaired.
May have early impairment.
Often impaired.
URINALYSIS
Usually normal. May show mild abnormals.
Often negative; may show mild albuminuria and cylindruria.
Albumin casts and occasionally microscopic erythrocytes.
Albumin casts and often microscopic erythrocytes.
Albumin casts, erythrocytes and even gross hematuria.
Polyuria and albumin casts, erythrocytes.
CHANGES IN Muscle Arterioles In Terms of Lumen to Wall Ratio Mean Normal Ratio=2.0
Ratio 1.5 — 1.8 Mean 1.7
Ratio 1.1 — 1.9 Mean 1.3
Ratio 1.0 — 2.0 Mean 1.3
Ratio 0.9 — 1.4 Mean 1.1
Ratio 0.8 — 1.4 Mean 1.5
Ratio 1.2 — 1.9 Mean 1.6
RENAL FUNCTION
Satisfactory
Satisfactory
Often, adequate. Sometimes early insufficiency.
Maybe adequate. Insufficient in late stages.
Often adequate. Sometimes early insufficiency.
Often severe insufficiency with increased urea and creatinin in blood.
PROGNOSIS
30 per cent
Mortality in 4 year period.
31 per cent
Mortality in 4 year period.
65 per cent
Mortality in 4 year period
93 per cent
Mortality in 4 year period
57 per cent
Mortality in 4 year period
77 per cent
Mortality in 4 year period.

Figure 14. Part of the Exhibit shown at the annual meeting of the American Medical Association at Cleveland, Ohio, June 11 to 15, 1934, by Drs. Norman M. Keith, Henry P. Wagener, and Nelson W. Barker, of the Mayo Foundation, Rochester, Minn.

from the use of the ophthalmoscope, sphygmomanometer and microscope and from special studies of the heart. To the well informed, the fundus of the eye reveals much of fundamental importance. From the standpoint of diagnosis it is the polished surface that takes the finger print of disease and from the prognostic viewpoint the wall in Belshazzar's palace on which is written the fate of the patient. Cardiac studies are also of the greatest significance since in most instances the heart fails before the kidneys.

Some Side Lights on Hypertension. Little is known concerning the cause of hypertension yet some light is being shed on its pathogenesis.

TREATMENT BY RADIATION OF PITUITARY AND ADRENALS (HUTTON)

Adequately treated and followed		261
Improved	180	68.9%
Relapsed	27	10.3%
Remaining improved	153	58.6%
Unimproved	81	31. %
Insufficient treatment		55
Can't be followed		27
		343

Contributions are appearing from the wards and from experimental studies in the fields of physiology and chemistry which have fundamental bearing on the problem of hypertension. The existence of a prehypertensive state has been revealed by the late Dr. George Brown by the use of his so-called "cold test." Much hope has surrounded the possibilities of relief from hypertension through sympathectomy. This movement was inaugurated some years ago by me in requesting Dr. Adson to remove the 2nd, 3rd and 4th sympathetic ganglia bilaterally in one of my cases of malignant hypertension. The drop in blood pressure following the removal of each ganglion was striking. The immediate clinical effects were excellent. However, the thought back of the suggestion was not the cure of hypertension, but rather the introduction of a safety valve effect; protection for the vital centers in times of stress by the relatively easy dilatation of the vessels of the lower extremities deprived of their vasoconstrictors. Much excellent work has been done in this field by Adson, Craig, Heuer, Peet and others. Many patients have been helped materially. But their work has also served to demonstrate that other and still more important factors are playing a major role in the pathogenesis of hypertension.

In this connection I wish also to call your attention to some most important clinical studies that are being carried out by your President, Dr. James H. Hutton. Some few years ago he advocated the simultaneous irradiation of adrenal and pituitary glands in the attempt to control the level of the blood pressure in hypertension. In a series of some hundreds of patients his results have been good in a high percentage of cases. (Table 15.) Having followed his technique in a small number of cases Dr. B. Widmann and I have also obtained results in many cases which are most gratifying both to us and our patients. In fact so favorable has the impression been that we feel that this simple measure should be tried in the majority of cases before resorting to the more radical surgical measures.

The most significant advances in our knowledge of hypertension have come from the investigations of Goldblatt and his collaborators. They may be tabulated as follows: (Table 16.)

Thus through continuous, controlled renal ischaemia, hypertension and many of its clinical manifestations have been reproduced experimentally, its pathogenesis has been somewhat clarified and the possibilities revealed of rational approach to treatment. Thus we are brought a step nearer to an understanding of the problem of hypertension. However, we have yet to learn the cause of underlying vascular changes which lead to the renal ischaemia and in turn to hypertension.

In this connection and largely as the result of Goldblatt's experiments, attention is turning again to the kidney itself. The experiments of Tigerstedt and Bergman of forty years ago are recalled, in which they obtained from the kidney a substance capable of increasing blood pressure which they called renin. This substance was said to raise blood pressure only in the species from which the kidney and renin were derived, i.e., that the effect was specific to the species. This has been disproved recently by the work of Landis which indicates effective pressure effects from renin of a heterologous origin.

Purification of renin and its biological assay has been carried out by Professor W. W. Swingle of Princeton. He has found that the pig's kidney represents the best source of renin. It is a globulin by nature. He has purified the globulin to the extent that in the dog 0.1 mg. given intravenously raises mean blood pressure 41 mm. Hg. and 0.4 mg. raises it to 112 mm. Hg. He has expressed its potency in terms of units—a renin unit consisting of the amount of renin necessary, when administered intravenously, to raise the mean pressure of a dog 40

EXPERIMENTAL HYPERTENSION THROUGH RENAL ISCHAEMIA CONTRIBUTIONS OF GOLDBLATT AND COLLABORATORS

1. Renal artery clamps and technic of application.
2. Bi or unilateral renal ischaemia induces hypertension.
3. Hypertension in dogs—5 years duration—in monkeys.
4. Degree of ischaemia determine type—benign or malignant.
5. Hypertensive eye ground changes and uraemia reproduced.
6. Hypertension—unaffected by a—sympathectomy; b—neurectomy; c—carotid sinus extirpation and d—hypophysectomy.
7. Aortic clamp just above renal arteries—same result.
8. Ischaemic hypertension—humeral in origin.
9. Nature of agent unknown—adrenals under suspicion.

Figure 14. Part of the Exhibit shown at the annual meeting of the American Medical Association at Cleveland, Ohio, June 11 to 15, 1934, by Drs. Norman M. Keith, Henry P. Wagener, and Nelson W. Barker, of the Mayo Foundation, Rochester, Minn.

mm. Hg. Renin is relatively non-toxic, 35 mg. intravenously proving innocuous to dogs except so far as increase in blood pressure is concerned. The rise in blood pressure is rather rapid in onset and persists over a period of hours.

Evidence is now accumulating to indicate that there exist accelerators and inhibitors of renin. Page has discovered a renin activator which is water and alcohol soluble which he calls "angiotonin," and Harrison, Grollman and Williams have found in the kidney a substance which they designate the "anti-pressor substance" which diminishes the pressor effect of renin. Thus we are launched on a new approach to the study of hypertension.

In conclusion may I state that we as practitioners are beginning to assume our true responsibility for our own intensive investigation of our clinical cases in this field. This is resulting in deeper insight into and better management of cardiovascular-renal disease. We are learning much of the role played by the kidney in this important combination of diseases. This appears easier of understanding today if we refer back to the work and especially the drawings of Bowman of a century ago, and to the methods employed by Bright in the clinical investigation of disease.

1520 Spruce St.

HOW THE POSTGRADUATE SERVICES MAY BE IMPROVED

HAROLD M. CAMP, M. D.
MONMOUTH, ILLINOIS

Nearly forty years ago the Illinois State Medical Society developed a Speakers' Bureau, listing men in all branches of medicine who were willing to go before county medical societies in various parts of the state to appear on their regular programs. From the records of the society, we learn that for two or three years there were discussions before the House of Delegates deploring the fact that so many members of this Society were unable to leave their work and go to some metropolitan center for intensive postgraduate work. They agreed that a plan to take men into the various counties would aid materially in solving this problem.

The original list of speakers contained the names of many physicians from all parts of the state, and an effort was made to publicize this fact in the ILLINOIS MEDICAL JOURNAL and from the office of the State Society Secretary. Unfortunately at that time they did not realize that to carry on such a program it would have to be handled by a separate committee or group outside the Secretary's office, and consequently the results were not nearly what had been expected. Within a short time this plan was abandoned.

At the annual meeting of this Society in 1923, a plan was developed which resulted in the formation of our Educational Committee. Donations were received from the membership to start the work which primarily was directed toward the building up of public opinion relative to the work of the medical profession. A Speakers' Bureau was established which has been enlarged from time to time, and many speakers appeared by invitation before hundreds of lay groups in every section of Illinois.

A few years later, our present President, Dr. James H. Hutton, thought that the Committee should serve another group, namely, the medical profession itself. So the Scientific Service Committee was established for the purpose of aiding county medical societies in arranging their programs, procuring speakers and later, in publicizing the meetings. Unfortunately many members of this Society have not thoroughly appreciated this work and have not known of all the services which have been available.

The resolution approved by the House of Delegates a year ago resulted in the selection of a Postgraduate Committee to study the plans now in operation in other state medical societies, and to develop a plan whereby postgraduate services could be improved within this Society. Dr. Robert S. Berghoff has given complete information relative to the activities of this committee, and the four test postgraduate conferences which have been held in recent months.

At the first Conference those physicians who were present were thoroughly agreed that this type of service was desirable, and they hoped it would be continued. At the second meeting, as another test, the committee agreed to send a mimeographed copy of each paper in full or in abstract to all physicians who registered. This plan proved to be so satisfactory that it was continued for the two later meetings.

Secretaries' Conference, May 21, 1940.

This Society has invariably been opposed to accepting Federal or State funds to maintain any of its activities, and has felt that the Society should finance all of its projects and have complete control of them. We learned during the investigations which were made during the past year that many state medical societies do accept Federal funds and their programs are, to a certain extent at least, supervised by Federal or State departments. Through the efforts of our Maternal Welfare Committee many interesting programs have been conducted in all parts of the state on Maternal and Child Welfare, and these have been under complete medical society supervision.

Much information has been received from state medical societies relative to their respective programs for postgraduate service as a society function. In one state they have the same program given in various parts of the state in turn, and issue certificates to physicians attending the meetings. If a physician is unable to attend the meeting in his area, he is permitted to attend elsewhere to get the same program and full credit is given under these conditions. Our committee believes that the success of a meeting is dependent on what the physicians get from attending rather than the procuring of "credits" or "certificates," and our efforts have been more along the line of presenting the type of programs which the physicians in that particular area desire, for they have the opportunity of assisting in the selection of both the speakers and the subjects.

In some states receiving Federal and State aid for carrying out their respective postgraduate programs, one specialist in obstetrics or pediatrics is employed to give the same program in various parts of the state on the one general subject for a period of one year, then the second year, taking another subject for a similar series of talks.

It has been the opinion of our committee that it is much better to have a diversified program which will be of general interest covering the more important subjects in present day medical practice. We have had hundreds of talks given in all parts of Illinois on obstetrical and pediatric subjects under the auspices of our own organization, and although we believe these are all equally important, we do not believe a postgraduate program on a state-wide basis should be so limited in its scope.

During a period of some six months, four experimental postgraduate conferences have been held in as many different parts of our state. The attendance at these four meetings was approximately 800, and we failed to hear any of the men object to the type of program or subjects discussed at any of these meetings. When those locally responsible for the selection of speakers and subjects, asked for certain men to make the presentations, the Scientific Service Committee endeavored to procure the speaker desired, and in several instances, it was men from adjoining states.

The report of the Chairman of the Scientific Service Committee will be presented before the House of Delegates at this annual meeting, and with it a supplementary report is to be given for the Postgraduate Committee according to the orders given a year ago by the House of Delegates. If the House of Delegates desires to do so, it may recommend that more postgraduate conferences be arranged for next year. It will be possible to arrange these schedules so that physicians in any part of the state will be able to travel a reasonable distance, and attend several of the conferences during the year.

When county medical societies desire individual programs and prefer to invite physicians from adjoining counties, the Committees will cooperate in every way possible and can schedule as many speakers as may be desired. When it is desired, special diagnostic clinics can be arranged as a part of either the district postgraduate conference or individual county society program. Heart clinics have been conducted in various parts of the state with the local physicians bringing in unusual types of heart cases for the clinic. Crippled children can be brought in limited numbers for an orthopedic clinic. Pediatric clinics can be arranged so that the children may be brought in for diagnosis or other consideration.

Dermatological clinics, or if desired, a tumor clinic, can be added as a part of the program at these conferences.

One of the highly interesting types of program which can be well scheduled, is a pathological conference where an internist can discuss the medical side, the surgeon, the surgical side, perhaps a radiologist to give the roentgenographic aspects of the subject, then the pathologist with his interesting specimens may conclude the symposium. Conferences of this type have been

scheduled in many counties, and they make an interesting type of program for either a district or local meeting. The pathologists have held a special meeting at this annual meeting for the first time this morning, and they desire to continue having a similar program each year to be scheduled as a special meeting to be held on Tuesday morning. This is not intended to be a meeting for pathologists, but one for the practicing physician with the program presented by the pathologists.

The program for this annual meeting is in line with our modern ideas concerning postgraduate education as there are more joint meetings of all sections with well balanced programs intended especially to benefit the general practitioner. The stag which has been held for so many years on Tuesday evening, has been replaced by a joint session with four speakers. The annual meetings can be arranged in the future to give the man in general practice more papers of interest instead of having highly technical papers scheduled which are of interest to only a limited group of physicians.

The Scientific Service Committee of this Society, now servicing nearly every county in the state, can send out more speakers to county societies, for tri-county, or district meetings, and the local officials arranging the meeting may select both the speakers and the subjects. The recently arranged list of speakers and subjects now available through the Educational and Scientific Service Committees, has made it much easier for the Program Chairmen to get what they desire for meetings.

The four experimental postgraduate conferences have been arranged through the facilities of the Scientific Service Committee, and conducted under the auspices of the host county society, and at the expense of the State Medical Society through a special appropriation granted by the Council. It was the desire of the Scientific Service and Postgraduate Committees in arranging these meetings, to give the members exactly the type of program they desired, and the results were most satisfactory.

On Thursday morning when the House of Delegates of our Society holds its last meeting, all reports which have been referred to Reference Committees for study, will be returned to the House for final action upon the recommendations of the Committees. If this House of Delegates desires to have these postgraduate conferences continued during the next fiscal year, they will recommend that this be done, and the matter will be referred to the Council for final action, on account of the fact that special appropriations must be made for this purpose.

The Secretaries of the several county medical societies will have an important duty to perform in connection with local arrangements for the proposed conferences, and if it is desired by this group, a resolution may be submitted to the proper Reference Committee, requesting that a larger series of these conferences be scheduled for the next year. A committee can be selected from this group if it is desired, to call upon the Reference Committee when the report of the Postgraduate Committee is scheduled for hearing, to give reasons for their approval of the suggestion. The Reference Committee, which is scheduled as Reference Committee "A", to receive reports of Council Committees, will welcome any suggestions this group of Secretaries, or any other group or any individual members may give them, and it will enable them to submit a better report to the House of Delegates at the closing meeting.

We have ten downstate Councilor Districts, and it is possible to have at least one postgraduate conference scheduled in each of these districts during the coming year. This would give an opportunity for many physicians to attend several of these sessions. The county societies in these districts should suggest the type of program they desire and also the speakers they desire to have present the various subjects. To make the program more diversified, it would be easy to have several clinics during these conferences. An hour could be devoted to the conducting of a heart, pediatric, dermatologic, orthopedic, or some other type of clinic, as may be desired. A pathological conference with a team of perhaps three can be scheduled if desired.

Short refresher courses on obstetrical and pediatrical subjects have been arranged in some of our medical schools during the summer for several years, and have become quite popular. It may be possible to have other subjects added to this list, and have same given over a short period of time during the summer months.

As each of you know, we publish each year a handbook for the members of the House of Delegates of this Society, in which is found the Annual Reports of officers, Councilors, and Chairmen of the various committees. You will notice

on reading the reports to be presented before the House of Delegates this year, that several committees recommend that each county society have one meeting each year devoted to papers on cancer, tuberculosis, obstetrical and pediatrical subjects, industrial health, physical therapy, and several other similar subjects. Unfortunately the average county society does not meet often enough to have programs such as are suggested in these reports.

These subjects are all of considerable importance to every practitioner of medicine, and each of them could be considered during the year as a part of a well balanced postgraduate conference program.

The Scientific Service Committee and other committees interested in the promotion of postgraduate activities within this society, will welcome suggestions from county society officials. If this group desires to have a series of postgraduate conferences conducted during the next year, they should give this information to the House of Delegates at the first Centennial Meeting of the Illinois State Medical Society.

VITAMIN K

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The sobering influence of time is a fine moderator of opinion. One should not be too hasty in accepting a new therapy, nor should rigid opinions preclude one from using any which offers help in the prevention or control of disease.

I first became deeply interested in this subject when Ivy, Shapiro, Melnick and I began a study of the value of viosterol and bile in the treatment of the bleeding tendency in jaundiced patients. The results of this study were published in 1935[1] and showed that a very favorable influence was exerted by the administration of these substances. The value of this method was further substantiated by the work of others.[2, 3] While it is true that the theory upon which this therapy was predicated was not in consonance with the facts now at hand, it is none the less interesting to call attention to the relationship which that work bears to the present discussion.

The bleeding tendency in jaundiced patients is due to a defective clotting mechanism[4] brought about by a reduction in the circulating prothrombin. Prothrombin is a complex globulin which is contributed to the blood by the liver. It must be present in sufficient concentration to react with the other elements of the blood to insure a stable clot. When its concentration level is below twenty per cent. of normal a defective clotting mechanism ensues.[5] It has been shown that certain substances which are absorbed from the intestinal tract speedily augment the prothrombin content of the blood. It is with these precursors of prothrombin that this discussion is concerned. The term vitamin K is used to designate this general group. The surgeon is concerned essentially with those pathologic physiological processes which disturb the normal intake, elaboration, assimilation and synthesis of the precursors of prothrombin.

The most important surgical conditions which disturb prothrombin levels in the blood are obstructive jaundice and biliary fistulae. In biliary fistulae the diversion of bile from the intestinal tract must be approximately eighty per cent. of normal before it seriously interferes with normal intestinal physiology. In obstructive jaundice the harmful effects are exerted directly in the liver and the blood stream and indirectly in the ailmentary canal. In the almentary canal the failure of bile to carry out its normal functions, when deposited there through the ampulla of Vater, is responsible.

It has been shown by repeated observations that the prothrombin level of the blood is very stable in health but is subject to marked variations in disease. It must be borne in mind that many conditions may act to produce a prothrombin deficiency. The precursors, or as Quick has so aptly called them "the building stones," of prothrombin are ingredients of a normal diet. These are absorbed from the intestinal tract and carried to the liver where they are synthesized into the complex molecule of prothrombin. Bile plays an important part in facilitating their absorption because they act like fats and are fat soluble. The exact role of the bacterial flora of the intestine in the elaboration of vitamin K fractions is not thoroughly understood but it has been shown that the common organisms such as the bacillus coli communis, the bacillus cereus

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and the bacillus subtilis are capable of synthesizing a fat soluble antihemorrhagic factor which they do not release or excrete into the media on which they grow. Many other bacteria do not seem to possess this activity. Micro-organisms of the mold, yeast or fungus type are generally inactive. It seems probable that a normal intestinal bacterial flora is important in maintaining an available supply of vitamin K. The condition of the intestinal mucous membrane must be such that assimilation can take place. The addition of vitamin K to prothrombin deficient blood in vitro does not facilitate its clotting. It would seem from this that the metabolic activity of the liver is the essential step before it becomes physiologically active.

The liver is therefore the center about which the whole process revolves. The degree of liver damage is the measure of defective prothrombin synthesis. In obstructive jaundice the liver damage will depend to a considerable extent on the liver reserve which was present before the immediate crisis developed. The rate of exhaustion of this reserve will depend on the character and the extent of the immediate crisis.

My observations compare very closely to those of other clinicians who find that the bleeding tendency is most marked in patients with an icterus index above 50. There is an increasing tendency to hemorrhage as the index mounts. The duration as well as the severity of the jaundice seems to bear a very close relationship to bleeding tendency. An almost universal reduction to dangerous prothrombin levels is found in patients whose icterus indices are above 50 and in whom the condition has existed over 60 days.

In the face of existing bleeding there is no question but that transfusions must be resorted to and repeated as often as necessary, or until the bleeding is controlled and the crisis past. It is unquestionably true that the prothrombin level of the blood can be maintained above the critical level by daily transfusion. It is noteworthy that blood which has been preserved for any length of time, the so-called "bank blood," loses its prothrombin content and should not be used. Citrated blood seems to lose none of its efficiency. In no instance should blood be unduly exposed to the light during administration. It is suggested that the prothrombin content of drawn blood is profoundly affected by light. During active bleeding the gastro-intestinal tract is frequently the site of hemorrhage into its lumen and therefore attempts at peroral administration of bile and vitamin K would meet with decided physical handicaps to absorption. If, as is freely predicted, a water soluble active substance which will be capable of facile prothrombin formation can be made, then we may find ourselves with a more potent therapy for the emergency case.

As a prophylactic measure, there can be no question but that every effort must be made to protect the jaundiced patient against the bleeding tendency. Every jaundiced patient should have an Ivy bleeding time taken and when possible a prothrombin clotting time determination. Even when these are not unusually prolonged antihemorrhagic therapy should be begun and continued until all danger is past. It is impossible to maintain a satisfactory prothrombin level if the liver has been damaged to the point where it is incapable of synthesizing those precursors which are available. It is very important that those who first see jaundiced patients recognize the necessity of fortifying and protecting the liver in the very early stages of the disease. The value of glucose administration in isotonic solution cannot be overemphasized.

In conclusion, it should be stressed that the bleeding tendency in the jaundiced patient is a real threat to his life. The earlier the prophylactic administration of antihemorrhagic factors the greater will be the effects and the better the results. In addition to vitamin K and bile therapy one should not neglect the supportive and rehabilitative measures which may be of equal importance. Glucose therapy, calcium administration and the use of vitamins A and C may play an important role in the patient's ability to combat the disease.

No single natural concentrate or synthetic product can be recommended at this time for they are changing almost as rapidly as the map of Europe. The production of a more readily assimilable product capable of intravenous administration will more nearly meet our needs and probably can be expected withn a short time.

CONCLUSIONS

1. Severe jaundice of the obstructive type is frequently accompanied by a tendency to hemorrhage from the skin and mucous membranes.

2. Diminished prothrombin in the blood

probably favors the bleeding tendency by interfering with the normal clotting.

3. The administration of prothrombin precursors is our best method of combating the hemorrhage tendency.

4. Active bleeding is probably best treated by repeated transfusions of fresh whole blood.

5. Every effort should be made to fortify the liver against the effects of obstructed bile flow.

6. The early release of obstructions in the biliary tract is emphasized.

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ON ANTITUBERCULOSIS VACCINATION BY MEANS OF BCG INTRODUCED INTO THE ORGANISM BY CUTANEOUS MULTIPLE PUNCTURES (METHOD OF S. R. ROSENTHAL) OR BY SCARIFICATIONS OF THE SKIN

L. NEGRE AND J. BRETEY

S. R. Rosenthal has recently discovered a method of vaccination against tuberculosis by means of BCG introduced into the body by cutaneous multiple punctures, which seems to us warrants the attention of doctors because of its facility of application, its complete inocuousness, and rapidity and regularity of appearance of a positive skin reaction to tuberculin.

In vaccinating guinea pigs, S. R. Rosenthal placed on the shaved skin, on each side of the vertebral column, one drop of a suspension of BCG (same strength as is used orally in the vaccination of new-born infants). He then introduced the point of a needle through the drop and pressed moderately to puncture the skin, making 30 such punctures through each drop. The needle was held tangentially to the skin. After a delay of from 6 to 15 days, small nodules, which did not surpass a pin-head in size, could be palpated at the site of inoculation. They disappeared in 2 weeks without suppuration and without leaving a scar. Histologic examination revealed (in the most superficial layers of the skin) typical tubercles with numerous giant cells of the Langhan's type, but the formation of pus or blocking of the lymphatic channels was never seen.

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Translation of a paper which appeared in *La Presse Medicale*, No. 5, January, 1940, page 49.

Rosenthal observed that this last process was entirely different from that noted at the site of the intradermal injection of a single dose of BCG. The latter induced the appearance of lesions in the reticular or even deeper layers of the skin, where necrosis, abscesses and obstruction of the subcutaneous lymph channels were observed. Giant cells were rare. S. R. Rosenthal proved that guinea pigs thus vaccinated and tested at regular intervals by the intradermal injection of .1 to .2 c.c. of standard tuberculin, diluted 1 to 10, reacted positively in the proportion of 58.3 per cent. by the 8th day, 87.3 per cent after 4 weeks, and 100 per cent. after 9 months. These animals still reacted to tuberculin after 3 years.

We have repeated the experiments of S. R. Rosenthal and have expanded them in trying to corroborate the degree of resistance, after the appearance of allergy, to a virulent infection of the animals thus vaccinated. We have followed exactly the technique of S. R. Rosenthal, but in order to limit the role played by the number of portals of entry of BCG, we made 30 punctures through one drop of the suspension of BCG in one group of guinea pigs, and 60 punctures through 2 drops of the same suspension in another group. The allergic reaction of these animals was studied at different intervals after the vaccination by the intradermal injection of .1 cc. of bovine tuberculin diluted to 1 to 20. In the group of guinea pigs vaccinated by 30 punctures, 47 per cent. reacted positively to tuberculin at the end of 1 week, and 63 per cent. at the end of one month. In the group of guinea pigs vaccinated with 60 punctures, 54.5 per cent. of the animals were allergic after 8 days, and 85.7 per cent. at the end of one month. Doubling the number of punctures augmented appreciably the proportion of the positive reactors to tuberculin, especially after a delay of one month.

The 2 groups of guinea pigs vaccinated with BCG by 30 and 60 punctures which reacted positively to tuberculin after one month, were injected subcutaneously with 1/1000th mg. of a very virulent human strain of tubercle bacilli. In sacrificing these animals at different intervals, we established that the vaccinated animals presented a very sharp retardation in the evolution of their lesions as compared with the controls. Thus, 2 animals killed after a delay of 4

weeks following the virulent reinfection presented only hypertropic inguinal and sublumbar lymph nodes. In most of them the spleen did not show any tubercles, while the latter were already numerous in the controls. At the end of 6 weeks, all the organs (spleen, liver, lungs) of the guinea pigs not immunized were permeated with numerous tubercles, while the organs of the vaccinated guinea pigs were in general free of lesions and their spleens rarely showed granulomas.

These experiments showed that the assertions of S. R. Rosenthal with regard to the rapidity and regularity of the appearance of the allergy were perfectly exact, and *established, moreover, that the guinea pigs thus vaccinated acquired a marked resistance to an infection produced by virulent tubercle bacilli.*

EXPERIMENTAL STUDY OF THE BCG VACCINATION BY CUTANEOUS SCARIFICATION

These points once established, we wished to determine if a modification of the above technique, namely a scarification of the skin, could be used as a means of vaccination.

To this end we adopted the following technique. One drop of the suspension of BCG containing 5 mg. per c.c. was deposited on the shaved skin of the lumbo-sacral region of the guinea pig, after cleansing the surface with ether, 5 to 6 scarifications 1 cm. in length were made with a vaccinating needle, avoiding too much bleeding. At the end of one week, infiltration appeared at the site of scarification. No suppuration or hypertrophy of the adjacent lymph nodes were manifested. Three or four weeks following this procedure, the skin had returned to normal without scars. In the guinea pigs thus vaccinated, without eliminating cachetic animals which reacted badly, we obtained results as follows: 40 per cent. positive at the end of 8 days, and 80 per cent. at the end of one month. From the point of view of the production of allergy, scarifications have given results almost equivalent to those obtained by multiple puncture.

The animals which reacted positively to tuberculin were tested with virulent tubercle bacilli in the same manner as in the preceding experiment. The guinea pigs vaccinated by the scarification method presented vis-a-vis to the testing infection a resistance similar to that presented by those vaccinated by the multiple puncture method. These methods were characterized by the precocity of the appearance of the allergy, since at the end of 8 days after the vaccination, 50 per cent. of the animals and sometimes more, reacted positively to tuberculin, and practically 100 per cent. after one month. As anticipated, the immunity proceeded alongside with the sensitivity, and all of our allergic animals infected one month after vaccination presented a very marked antituberculous resistance. According to the experiments which are now in progress, it seems that immunization is produced before this delay.

It appears, then, from these experiments, that in the guinea pig the multiple puncture method of S. R. Rosenthal and the scarification method present a certain advantage over other parenteral methods, as far as the precocity of the appearance of the allergy is concerned. These methods seem to give the guinea pig the same antituberculous resistance as the subcutaneous or intradermal injection of BCG.

FIRST ATTEMPTS OF APPLICATION OF THESE METHODS IN HUMAN CLINIC

Attempts at human vaccination by these methods were first made by S. R. Rosenthal and followed by B. Weill-Hallé. The results can only, for the moment, inform us as to the rapidity of the development of allergy. They have too recently been applied to confirm, on a clinical basis, what we have already observed of the immunity they confer on animals. At the Tice Laboratories in the Cook County Hospital in Chicago, S. R. Rosenthal vaccinated with BCG, infants of a non-tuberculous environment. The mother and other members of the child's family were first examined clinically and by x-ray. Vaccinations were done between the fifth and seventh day after birth. S. R. Rosenthal placed one drop of the suspension of BCG (the same strength as used in oral administration) on the arm of the infant and made 35 punctures of the skin through this drop, holding the point of the needle tangentially to the skin. The surface of the skin which received these punctures presented only a local reaction, consisting of pinpoint to pin-head sized nodules. Enlargement of the draining lymph nodes was not noted consistently. Rarely, small scars not exceeding pin head in size were noted after 3 months.

Another group of infants, under the same conditions, were vaccinated by the intradermal method, and still another group were kept as controls. All of the children were followed by a visiting nurse and tuberculin tested at the end of 3 months with .1 cc. of a 1 to 1,000 dilution of old tuberculin, and at the end of one year with a dilution of 1 to 500 dilution of old tuberculin. These tests were read at the end of 48 hours.

In April, 1939, Rosenthal had done 317 vaccinations, 177 by his method and 140 by the intradermal method. 320 infants served as controls. After 3 months, 99.2 per cent. of the infants vaccinated by Rosenthal's method reacted positively, and 97.7 of those vaccinated by the intradermal method. None of the control infants reacted. At the end of one year, the children vaccinated by Rosenthal's and the intradermal methods reacted 100 per cent. to old tuberculin. In the control infants, the proportion of positive reactors was 1 per cent.

Modifying the technique of multiple punctures as described by S. R. Rosenthal to that of scarification, Weill-Halle has vaccinated since last February, 26 cases for the first time and 30 revaccinations by the following procedure. He placed 3 drops of the suspension of BCG (same strength as used in the oral vaccination of infants) on the skin of the arm 2 or 3 cms. away from each other, and effected across these drops a scarification in the form of a cross measuring 1 cm. in length. In addition, after waiting 1 or 2 minutes, he applied on the scarification site a compress of gauze impregnated with an emulsion of BCG. He covered this gauze with an oiled silk, and kept the dressing on for several hours. The course of the vaccinations was most simple. The trifling wound of the scarification disappeared after 2 to 3 days. After the 15th to the 25th day, depending on whether it was a primary or a secondary vaccination, traces of the scarification reappeared and the skin presented a minute elevated scar. This remained for several weeks and disappeared in 2 to 4 months after the vaccination without any lymph gland enlargement or general disturbance. The children thus vaccinated who reacted positively to tuberculin were tested by the method of von Pirquet or percutaneously. It is possible that in the course of our present experiments better results may be obtained by modifying the strength of the emulsion as well as by certain details of technique.

CONCLUSION

Our conclusions are based entirely, for the moment, on the experimental results obtained by S. R. Rosenthal and by us. The multiple puncture method and the scarification method appear to have practically equivalent effects. Their use does not necessitate a different suspension from that currently used orally, and their interest appears to reside in the following:

1. The rapidity of the appearance of the allergy and the immunity;

2. The degree of this immunity, which is at least equal to that conferred by the intradermal and subcutaneous injections of BCG;

3. These results were obtained without the risk of complications observed in other methods.

According to these preliminary experiments, it seems to us that practitioners have now at their disposal a new procedure of introducing BCG into the organism, which merits their attention because of its facility of application, its harmlessness and its rapidity of action. The experimental results are very convincing, and the clinical efficacy is still to be determined.

In the troubled period through which we are now passing, we will probably witness a great recurrence of tuberculosis, similar to that observed during the last war. It is well established that vaccination by BCG of new-born infants and non-allergic subjects permits us to strive efficaciously against the development of this disease. It appears to us, then, advantageous to draw to the attention of practitioners these new methods of antituberculous immunization, which act after the shortest time and with the greatest possibility of success, as judged from the experimental results.

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SOCIALIZED MEDICINE

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Progress is not automatic. The world grows better because there are high-minded souls who wish that it should, and because they will and dare to take the right steps to make it better. So we commemorate the efforts of those great pioneers of medicine, who felt that the scheme of human relationship was out of balance, and capitalizing the fellowship instinct and the altruistic desire to serve, inherent in most men, gave us organized medicine. To them we acknowledge a debt of gratitude.

The finest ideals will not propagate themselves. In organized medicine we have the happy combination of ideals plus organization. Individuals may worthily desire to serve and build, to imbibe deeply of friendliness, tolerance and understanding, but alone they fail to impress the armored hide of indifference, selfishness, hate and bigotry. But with an organization of men similarly imbued with and fortified by an exchange of ideas, mutual helpfulness, and a splendid association which marshals for him an array of leadership, experience, facts and literature, and binds all together in a perfect union, he becomes an integral part of a great altruistic force for human good.

The American Medical Association is a great body with maturing obligations and of recognized importance in the councils of the continent. We may be proud of the past but we grow with the years. We think of the fine and outstanding achievements of a glorious past, but we consecrate ourselves to a larger future of helpful service to humanity.

The United States owes an incalculable debt to the American Medical Association.

Whatever criticisms now may be aimed at it by the idealistically minded who are shocked at obvious imperfections in the medical services available to the people as a whole, the fact remains that the organized profession itself, voluntarily and from a sense of duty, is responsible for about everything "social" in the practice of the healing arts of today.

It found American medicine in a chaotic condition. There were essentially no minimum standards of education or of competence. In most States a few years as helper in a doctor's office or around a hospital and the passing of a written examination were sufficient to launch a man on the practice of medicine. The American Medical Association has worked ceaselessly for higher and higher minimum requirements. It has put low-grade medical schools out of business. It has made the acquiring of an M. D. degree and a State license to practice a major struggle for any man and a hopeless struggle for an individual of mediocre intelligence. The public now is absolutely assured that any man who has graduated from an American medical college and passed a State board examination is highly competent. It is reasonably assured that he is not lazy or careless, unless his personality undergoes a remarkable change after leaving college. And it is reasonably assured that he is not a scoundrel, for dishonesty hardly could get through the filter of present-day medical education, for which the American Medical Association is responsible.

It has waged a strenuous fight to eliminate quackery. Contemptuously defiant of slander and libel suits, it has mercilessly exposed the nonsense of healing cults and alcohol-and-water nostrums. It probably would have succeeded altogether were it not for the obstacles thrown in its way by politics.

Through its local units it has rigorously punished, by censure and by expulsion, violations of medical ethics.

It has waged an unceasing campaign, both nationally and through these same local units for the health education of the public. By cold experimentation in its laboratories it has established the values of new remedies. It has constantly encouraged medical research and has kept the entire profession aware of the latest advances in medical science.

This is not the time for didactic essays or ornate orations. In these days—to use the fine phrase, "the times that try men's souls"—the only thing that is valuable in speech is sincerity, and it is in that spirit I speak to you on "The Menace of State Medicine."

Two widely antagonistic forces are striving for dominance in America. On one side, is the desire and ambition of the individual to live his own life and carry his own responsibilities and secure the utmost mental and material development, while on the other is the ambition to have

the people subjected wholly to herd ideas whether advantageous or otherwise—with only an inner certitude that the way the herd is directed is also the best way. The contest is between individuality and regimentation, and while regimentation with its attendant oppression has secured high place among decadent nations of Europe it should be fought bitterly in America which has grown great through private initiative.

What is socialized medicine? Socialized medicine is a broad term. Anything is socialized which is supported by people as groups rather than as individuals. Given many different names, it is sometimes called state medicine, which indicates medical services furnished by government employes who are paid out of tax funds. When we think of socialized medicine we assume that it would cover everyone. In practice it does not work that way. Most medical plans cover only industrial workers. Independent workers, such as shopkeepers, professional men, and farmers are excluded. The reason for that is, that while it is easy to have the periodic contributions deducted from the worker's pay envelope, it is difficult to make regular collections from the self-employed. In Germany, where it has been in effect since 1883, only about 40 to 45 per cent of the population are covered. In Great Britain, where the practice began to be operative in 1911, 39 per cent are cared for.

We next come to the question of how much it would cost for the individual to be cared for under such method as this. The Michigan State Medical Society spent $15,000 in an investigation to obtain this information. For the purpose of administering the plan, the Michigan committee estimated that the average family consisted of 4.1 members and set $118 as the tentative fee for the average family whether it has two members or a dozen. Contrasted to that, the average cost per family today actually is $62.00 under our present system of medical care.

The Committee on the Costs of Medical Care which began its studies in 1928 and completed them in 1932 consisted of 48 members. They published 28 major reports and a number of miscellaneous pamphlets dealing with medical care and its costs. This committee showed that 90.2 per cent of the people who are ill at any one time receive medical care. The other 9.8 per cent is easily understood when you consider the fact that approximately 50 per cent of the illnesses in this country are common colds or other bronchial ailments, and you can readily understand that many people feel that they are not ill enough to need a doctor. Also, there is a certain proportion of persons, who for religious or other reasons, will not consult a doctor under any circumstances. Those who argue mostly for controlled medical care are largely the sociologists and the Socialists, and a scattering of promoters whose discontent obscures its appreciation of the value in the present system.

Recent investigations by the Bureau of Medical Economics of the American Medical Association proved conclusively that there are few, if any, in the United States really suffering from want of medical care. The mayors of cities of various sizes testified that there was no neglect of the poor because of their inability to pay. Those who argue that people are not taken care of ignore the fact that doctors always base their charges on the ability to pay and are even willing to carry this policy of rendering the service free of charge to those who need it.

It is significant that the Committee on the Costs of Medical Care made no attempt to show how many sick people sought and were denied medical care because of poverty. The reason is that there was practically none. J. Weston Walsh, in compiling a handbook on state medicine, addressed questionnaires to physicians and public-health officials the country over. The reports were practically unanimous that few, if any, are denied proper care.

It might be asked: Is such free service as is now available degrading to the poor? We have every sympathy for the poor who wish to maintain their self-respect. Yet in viewing this matter it is necessary to perceive a sense of proportion. No one has yet suggested that the state take over the farms and factories and provide everybody with food and clothing merely to save those unable to pay from feeling that they are accepting charity. A little sympathy for the doctor may not be out of place. Nobody ever heard of a grocer reducing prices to those unable to pay, but the doctor does. The trouble is that many people regard a sickness as an accident for which they are not to blame, and therefore they do not feel the obligation of medical bills as much as the obligation of installments on the radio. They should be educated to understand that illness is to be expected. The costs should

be prepared for and, in justice, paid as promptly as other bills. The Michigan State Medical Society found that the annual expenditure of the average farmer's family for physician's care (considering a family as 4.1 people) is $62. The family tobacco bill is more than the farmer's family doctor bill. Twice as much is spent for candy as for hospitals. Twice as much is spent for cosmetics as for nursing.

When it comes to costs, let us look at a comparison in the United States Army. The Army is a picked group. Each man is selected because he is healthy. We would naturally expect this group to have the smallest per capita medical costs in the world, but John R. Neal, M. D., writing in *Clinical Medicine and Surgery,* declares that the Army medical system costs just double the average per capita medical costs for the United States. This is confirmed by the commitee on costs of medical care itself when it found that Army medicine at Fort Benning cost just $50.67 a person, or the equivalent of $200 a year for the average family of four.

America is now leading all other nations in the extent and quality of her medical research. Perhaps this is due to the fact that physicians in other countries have not the time to carry on such work. The profession is not fighting socialized medicine to preserve its own existence. It is fighting to keep the hands of the politicians from controlling the practice of medicine to the detriment of the health of the American people. It is fighting to prevent a gross bureaucracy from wedging itself between doctor and patient. The State cannot manage without commanding, and it will happen, therefore, when the State assumes authority that our profession will fall under the thumb of the politician. The dictation to the doctor by lay boards in certain medical institutions is bad enough, but such dictation becomes insufferable when exercised by self-seeking ward heelers and ruthless political organizations.

Medical care has been classed as a necessity by the national government. Not so very many services are given that high rating, and all services so rated cost a good deal of money. We hear so much distressful talk about the suffering of the under-privileged and the indigent because of the high cost of medical care. These two unfortunate classes have but little money in any event, and there can be no doubt that they suffer from lack of some of the other necessities as well. Those who propose a change in the present system of practice make no provision whatsoever for these people; they are still left to the charity of the private practitioner of medicine, because the beneficiaries of state or socialized medicine must have steady jobs or some other effective means of contributing to the massive coffers required to pay the expenses of the system. So in discussing the cost of medical care we are justified in leaving out of consideration the underprivileged and the indigent, because they are now cared for without cost by the private practitioner. Leaving out of consideration these two classes the people of our nation are not so poverty stricken. Yet we are advised to turn from the system of private practice to some form of socialized or state medicine; to follow the lead of Germany and England. We crossed the ocean once to get away from the lead of European countries, and by adopting different customs and laws, built here the greatest civilization in history.

Germany has had State Medicine for more than fiftv years; it was introduced there by Bismarck as a political measure and not in response to public demand. It was introduced into England by Lloyd George as a political measure. Lloyd George promised the industrial workers that he would give them two dollars and a quarter in medical care and cash sick benefits for every dollar they contributed—the "something for nothing" that people have always sought after. No form of socialized or state medicine has ever been proposed seriously that did not offer its beneficiaries a good deal more than they paid for; this is probably the secret of its popularity. The various types of socialized medicine follow pretty well one pattern. The employee pays a certain percentage of his wage into a fund, the employer pays a certain percentage of his pay-roll into the same fund, and the balance is provided by general taxation. The fact that taxation provides a part of the money to defray the expense of socialized medicine places the system pretty well under the influence of politics, and the politician is seldom trained in medical matters.

The beeficiary of socialized or state medicine receives medical care—not complete, under most systems, and cash benefits when he is sick and not able to work. Since his doctor bill is always

paid, he is supposed to feel very free about consulting the doctor. That is one of the strong points urged in favor of the system. There may be some defects in that theory that do not become apparent until tried, but the point of interest is: Does socialized medicine provide better medical care than is provided by the system of private practice? If it does—if under it prevention of disease is better carried out, early diagnosis promoted, morbidity reduced, mortality from disease lessened and the span of life more rapidly lengthened—then it would be fair to conclude that our system of private practice should be relaced by one of the systems of socialized or state medicine.

It is conceded that the beneficiaries of socialized medicine consult the doctor in very large numbers. It is also conceded that the majority of them seem to have minor and imaginary ills, and that a surprisingly large percentage of them consider themselves too sick to work, and, therefore, entitled to draw sick benefits during their period of disability. The cash benefits that are paid during sickness sometimes make a severe strain on the treasury and lead to investigation by the business department of the system. That is a source of much trouble, not only to the doctor but to the person who feels entitled to the benefits. It is sometimes considered necessary to send out the consultant of the business department to see if the doctor in charge can be persuaded to cut off some of the beneficiaries and the business department usually has ways of doing this in spite of the judgment of the doctor in charge of the case.

State medicine goes in very strongly for record keeping. After the doctor has cleared his waiting room he must spend much time, as a rule, filling out the large number of blanks provided by the statistical department. This is the time which the doctor who practices private medicine usually devotes to reading his medical journals or his textbooks, which is one of the chief means of keeping up with advances in medicine.

In the United States, under private practice, the industrial worker loses from eight to thirteen days a year from sickness; in Germany, under socialized medicine, the industrial worker loses from fifteen to thirty days and he is supposed to be the chief beneficiary of socialized medicine.

Politics enters more or less, often more, into the management of socialized medicine. It does not promote the practice of preventive medicine. Its diagnostic service is inferior, and morbidity rate is greatly increased, and the mortality from nearly all of the important diseases is greater than under private practice.

Under the system of private practice as it exists in this country, there is one other feature which, in the opinion of most doctors and most patients, adds greatly to the service rendered—the personal relationship which exists between the doctor and the patient. It has become fixed in the customs of our people and it will continue until changed or destroyed by law. One well trained doctor may be as able as another to apply the truth of science in the treatment of disease, but times come in the life of each one of us when the cold facts of science do not avail. The personal side of the private practice of medicine, which has always played an important and comforting part, steps in at such times and renders a service which the people not only desire but demand. Sympathy, kindness, pity, and cheerful hope—no amount of scientific efficiency can take the place of these in the dark hours of sorrow and trouble so common in the experience of all. President Eliot of Harvard said: "In these intangible things are found the durable satisfactions of life; fame dies and honors perish, but loving kindness is immortal."

I would not belittle the importance of science in medicine—I bow in humble reverence before its beneficent power, nor would I magnify the personal element, yet many of us know from experience what comfort, hope and assurance the personality of a trusted physician may bring to the bedside of his patient.

Socialization tends to destroy personal service; it places all of the emphasis on the scientific side and while the scientific side is the greater, yet divorced from the personal element it is immeasurably weakened. Our system of private practice blends the two into one service, and thus the medical care received by the American people is the envy of the rest of the world. In no other nation has medicine wrought so well in bringing health and happiness, and length of days to the fleeting span of life.

Recently Dr. Frederick L. Hoffman, statistician for the Prudential Life Insurance Company of New York, and probably the outstanding medical statistician in the world, made

comparison of the mortality rate of some of the leading causes of death in England, which has a system of socialized medicine, with the mortality rate from the same diseases in the United States, which still has a system of private practice. I will mention quite briefly a few of these comparisons. In England the date rate from tuberculosis of the lungs is 63.5 per hundred thousand; in the United States it is 51.2. In England the death rate from cancer is 156.3 per hundred thousand; in the United States it is 106.3.

The complaint against the cost of medical care under our system of private practice made by the advocates of change is that it is too high. No one has ever argued that medical care is not expensive, and that it does not sometimes fall with more or les crushing effect upon the unfortunate —but, other misfortunes have a way of doing the same thing. It is rather certain that socialized medicine is able to distribute the cost of medical care in such a way that the burden is not so keenly felt by the beneficiary, but the cost to society, which is the true index of cost, is very much greater. The business set-up which looks after the distribution of medical care in socialized and state medicine, employs a great many people—inspectors, bookkeepers, supervisors, and so forth, and these must receive a living wage.

Take, for example, Germany. In 1935 there were 36,000 employees of the non-medical personnel, and only 30,000 doctors. The politicians are supposed in theory, to keep their hands off the business and professional set-up of socialized or state medicine, but where they vote a considerable part of the money to pay the expenses of the system, they are naturally interested in the management. Sometimes they take quite a bit of interest in it. In one European country, several thousand doctors have been deprived of the privilege of practicing medicine for the state because they incurred the displeasure of the political powers. Politics is intimately bound up with the administration of socialized medicine in all cases where the state is called upon to pay large sums of money for its support, and no one should expect it to be otherwise.

Government participation in medicine should be restricted instead of enlarged. Mindful of the medical profession's ageless and fruitful tradition of self-sacrificing service and of the American people's stake in personal freedom, government should refrain from competing in or monopolizing the field of medicine.

If the governmental agencies will keep hands off, the medical profession will work out the problem along lines based on American ideals.

The agitators for Federal medicine have not proved that there is any need for such legislation. Let them prove their case and not go off on a tangent, and then be compelled to beat a hasty and ignominous retreat.

The American people are a proud people and do not wish some political bureau to enter their home life, to administer their routine in illness, to invade their right of privacy. Federal medicine would be a tragedy for the American health record—the best in the world.

By means of a calm and dispassionate marshaling of facts, let us redouble our efforts to convince the public that socialized medicine is "poorhouse medicine."

AN EVALUATION OF THE ELECTROCARDIOGRAPH

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It is with great temerity that I title this paper "An Evaluation of the Electrocardiograph." I realize that there have been many such papers written by men who have been studying the subject for years, but I feel that a review of the things that we already know or think we know is often of great value. Some of the statements to follow are personal opinions and I shall welcome critical discussion of the entire paper.

As you know, the electrocardiograph is not a new instrument. It was developed in the form used at present by Einthoven in 1905. It was not, however, until Lewis popularized its use after 1915 that it created much attention. In the nineteen-twenties there was much dispute as to its value. Some of its proponents were making great claims for it and others of just as good reputation stated that it was of little or no value. In the last ten years its use has been more universal and its true place in the practice of medicine is appreciated. Like so many of the newer things in our profession it had the usual cycle of doubtful acceptance, then overenthusiastic claims, a reaction period when it was used little, and then a gradual return to use in its proper place. I well remember two of my teachers in

medical school, one stating that he was sorry he had invested in an electrocardiograph as it was of no use whatsoever to him, and the other man, just as competent, advising its use as a routine part of every examination.

The instrument itself is rather complicated and delicate in its construction, but it is based on a very simple principle. In reality, it is a very sensitive galvanometer similar in principle to that we all used in physics laboratories. The electrocardiograph is made in such a manner that it records on a permanent support, either film or paper, the amplitude and the direction in which an electric current is traveling. As you remember from your physiology all muscles when in a state of contraction become polarized and an electric current can be demonstrated to be generated during the contraction. The electrocardiograph is based on this fact. The tracing is merely the recorded evidence of the electrical changes in the heart muscle. This record is so made that the time relations of the various changes during individual heart contractions can be easily seen and the pattern of succeeding contractions compared with each other.

You may wonder why this restatement of very elementary facts. The answer is that without a realization of the basis of the instrument any statements made relative to the interpretation of its records may not be easily understood. I am concerned in this paper as much with what the electrocardiograph cannot do as with its positive virtues.

In general, there are two broad classes of heart diseases. Of course they overlap and coexist, but for the purpose of discussion, I am speaking of the disorders of rhythm first and the pathological changes in the myocardium second.

The first use, and for a time thought to be the only use, of the electrocardiograph was in the analysis of disorders of cardiac rhythm. It was found empirically that in most apparently normal individuals the cardiograph produced a tracing with certain characteristics which did not vary. *On the basis of many thousands of tracings on supposedly normal individuals an empirical normal tracing was established. This* tracing showed a definite time relation between *the component parts of the graph. Experiment* showed that each portion of the cardiac cycle had *a definite correlative and characteristic modification* of the base line in the cardiograph record. Therefore, irregularities in the mechanics of each heart beat could be analyzed. Cardiac arrhythmias can and should be diagnosed clincially as far as possible, but the court of last resort in their analysis has come to be the electrocardiograph. Frequently, an accurate diagnosis of the location and severity of an arrhythmia is indispensable in prognosis and treatment. A discussion of a few of the commoner arrhythmias may serve to emphasize this point.

By all odds the commonest arrhythmia we all see is sinus arrhythmia. The name means that the disordered rhythm is due to some interference with the rhythmic functioning of the sinus node and that the conduction mechanism within the heart is not damaged. Each beat is normal, but the initiation of the sequence of systoles is not. Frequently the arrhythmia is coincident with the respiratory cycle. It is important that we recognize a sinus tachycardia for what it is, as serious mistakes in treatment may be otherwise made. Sinus tachycardia usually accompanies all the infectious diseases or processes which elevate the basal metabolism. Digitalis formerly was used almost routinely to lower the excessive heart rate in these diseases, but in rheumatic fever the danger of producing heart block is great if not carefully administered[1] and in thyrotoxicosis an upset digestion as a result of digitalis therapy may defeat proper dietetic management. Sinus bradycardia on the other hand is usually of little significance except in that it is important to distinguish it from other more serious bradycardias. This can be done most positively by the electrocardiograph. Sinus arrhythmia has been spoken of as a sign of a healthy heart. Unquestionably it is seen more often in children and young adults than in any other group and has little significance.

The commonest arrhythmia which is of definite pathologic interest is auricular fibrillation. Lewis states that it constitutes 40 per cent of the disordered rhythms.[2] Most of us feel that we are competent to identify an auricular fibrillation *by clinical methods alone. In most cases* this is true, but clinically it is often confused *with auricular flutter with a variable conduction* block, ventricular tachycardia, and a sinus *rhythm plus frequent extrasystoles.* We might be embarrassed, to say the least, to give digitalis or quinidine to a patient with frequent extrasystoles thinking that we were treating an auric-

ular fibrillation, only to have the extrasystoles increased by our treatment. A ventricular tachycardia may be converted by quinidine into a ventricular fibrillation; in a questionable case the electrocardiograph will prevent such errors by demonstrating unquestionably the type of arrhythmia.

The next most common disorder of rhythm is the premature contraction or extrasystole, constituting according to Lewis, 35 per cent of cardiac irregularities when it arises from a single focus. We usually do not attach much importance to these but feel that an electrocardiogram should be made to be sure the extrasystoles do not arise from more than one focus. It should be pointed out that when the electrocardiograph shows frequent extrasystoles arising from multiple foci in the heart, we are dealing with a potentially very badly damaged heart. The chances are excellent that before long a more serious disorder of rhythm will occur. The condition has been called "chaotic heart" by Katz and in his experience means a shortening of life expectancy to not more than two years.[3].

About 15 per cent of the arrhythmias are made up of paroxysmal tachycardias, heart blocks of various degrees, flutter and the sinus arrhythmias. You are all familiar with the typical picture of paroxysmal tachycardia. Usually there is a clear-cut syndrome which can be recognized without laboratory methods. The electrocardiograph is of utmost value when the clinical method is uncertain and the diagnosis must be made for the sake of offering a correct prognosis. As you know, the prognosis of a paroxysmal tachycardia of ventricular origin is much less hopeful than that of an auricular tachycardia of auricular origin. In older people especially, the electrocardiograph should be used in all tachycardias that are in the least unusual, because it is a well-established fact that ventricular tachycardia is seen most often in association with coronary vascular disease.[4] Auricular tachycardia on the other hand, while causing much distress to the patient and his family usually does not alter the life expectancy and produces only the immediate morbidity of the attack. It is seen many times in hearts that appear to be completely normal. Auricular flutter with a high ventricular rate is often not of great significance in itself, but since it is assigned a place intermediated between auricular tachycardia and the development of auricular fibrillation, it is important to know the mechanism of the arrhythmia in order to establish a prognosis. This can be done most accurately by the electrocardiograph.

A diagnosis of heart block is often made on clinical grounds, particularly when the block is above the ventricles, but the clinical diagnosis of an intraventricular conduction block is difficult to make without graphic methods. A discussion of heart block is important for two reasons; one that conduction block may be produced by our most commonly used cardiac drug, digitalis; the other intraventricular block, is most often seen in advanced myocardial disease, usually on the basis of a coronary occlusion with myocardial infarction. This statement brings to mind a discussion that occurred not long ago regarding the electrocardiograph. An attending man asked me about a tracing and in conclusion asked "Does this patient need digitalis?" I replied that I did not have the slightest idea. The electrocardiograph will tell us nothing about the indications for digitalis, but it most definitely will tell us when the patient is receiving too much. The signs of digitalis intoxication seen on the electrocardiographic tracing are almost pathognomonic and can be seen before the clinical signs appear at all. The earliest sign of digitalis administration is a beginning of block of conduction either in the sinus node, or in the auriculoventricular node. Digitalis produces definite changes in the electrocardiographic tracing. These are sometimes so marked that they obscure changes produced by the heart itself. If you wish a true picture of the electrical activity of the heart and feel that the welfare of the patient does not demand digitalis, do not give it. If digitalis has been given we must realize that the tracing will be modified by it and our integration of the electrocardiographic interpretation into the clinical picture must be colored by this fact. Thus we see that the electrocardiograph is of value in the management of the digitalized patient.

Time will not permit the discussion of other forms of cardiac arrhythmias so I will pass on to another field in which the electrocardiograph is of great value, that is, the diagnosis of changes in the myocardium itself.

The myocardial change with which we are most concerned is that due to coronary occlusion. You are of course aware of the fact that coronary

occlusion is not a new disease but its definite recognition clinically does not extend back to more than twenty-five years ago with the work of Herrick. It was at this time that Lewis was experimenting with the electrocardiograph and felt that the instrument was of primary value in analyzing the cardiac rhythm. Probably one of the earliest electrocardiographs made showing the presence of coronary occlusion was made in the laboratory of Lewis in 1913. At that time he did not interpret it as an illustration of coronary occlusion, but rather as an instance of transient bundle branch block. Hermann and Ashmann have called attention to the fact that the electrocardiographic curves in this case now would be considered highly suggestive of coronary disease.[5] Further study of clinically demonstrated coronary occlusion showed that definite changes occurred in the tracing which were not present in the absence of clinical signs. Correlation of the electrocardiograph with autopsy findings finally demonstrated that the electrocardiograph was capable in the majority of instances of making a diagnosis of coronary occlusion with myocardial infarction with great accuracy. The work of Woolferth and Wood in the early 1930's using a new lead taken from the anterior chest wall added to the accuracy with which diagnosis could be made. Their method was particularly valuable in anterior infarctions which were not infrequently missed when using only the standard limb leads. Many modifications of this Lead IV were suggested and it was not until 1938 that a special committee of the American Heart Association reported on the technique which was the most accurate. By the use of standard limb leads and the chest lead approved by the American Heart Association, a uniformly accurate diagnosis of myocardial infarction was made.

Perhaps someone wonders why I did not say coronary occlusion, but rather myocardial infarction. If we review physiology a little, the answer becomes obvious. The electrocardiograph demonstrates changes in the electric current produced during the heart's contraction and relaxation, but nothing more. It can give us no direct measure of the competency of the coronary arteries. We know what the usual tracing looks like taken from a patient with coronary occlusion and myocardial infarction; we see definite changes. These changes are due to the difference in the character of the electrical currents produced by damaged or necrotic muscle from those produced by normal muscle. To be technically correct in interpreting electrocardiograms we should say "myocardial infarction," rather than "coronary occlusion." However, on the basis of statistical evidence from autopsies, we can say that in the absence of external trauma, myocardial infarction is most often due to coronary occlusion. When a coronary occlusion occurs the blood supply to the myocardium is cut off. If the occlusion is permanent (in the sense of lasting more than a few minutes) the same pathologic events occur as would occur in any muscle deprived of its blood supply: ischemia, followed by slowly developing necrosis. The electrocardiographic changes may not occur for several hours after the actual occlusion. They do not occur until the ability of this damaged muscle to produce normal electric currents becomes impaired. I emphasize this because time and again the clinician makes a diagnosis of coronary occlusion, has an electrocardiograph made immediately, gets a report which is essentially normal, and then either feels that his diagnosis is wrong or damns the instrument as unreliable. In a strongly suspected case of coronary occlusion supported by definite clinical data, repeat the electrocardiograph in 24 hours even if the first tracing is essentially negative. If the second tracing is still essentially normal, only then begin to doubt your diagnosis. "In acute coronary occlusion the electrocardiographic changes are almost always sufficiently characteristic to be practically diagnostic, both as to the presence of the lesion and as to its location."[7] Very rarely the electrocardiograph may present a picture of coronary occlusion when the lesion is a pulmonary embolism, a non-penetrating wound of the heart, or a pericardial effusion. These should not be confusing because they have definite clinical pictures to help differentiate them from coronary occlusion.

We hear much today of the term "coronary sclerosis." It is probably a good term, but I prefer not to use it in speaking of electrocardiographic changes. "Coronary insufficiency" probably is a better clinical description. It is possible to have a coronary sclerosis of somewhat marked degree and yet have no evidence of impairment of the heart's nutrition. You may have noticed in the reports of electrocardiograms,

the statement that probably there is present a coronary insufficiency. The word "probably" is definitely necessary in this statement because coronary sclerosis without thrombosis is apt to produce irregular electrocardiographic changes, involving any or all of the waves and complexes, depending on the extent of the cardiac muscle damage which resulted from the arterial disease. Such changes however, are not characteristic or diagnostic, since myocardial damage from any other cause may produce similar alterations. "Sometimes an entirely normal tracing is obtained in the presence of extensively diseased coronary arteries."[8] "If an infarction occurs due to an arteriosclerotic closure of the coronary arteries, the electrocardiographic changes may not develop for two or three days, whereas in the sudden occlusion they may develop in a few hours."[9]

In all cases of coronary occlusion the electrocardiogram should be repeated at frequent intervals. It has been shown that as the necrotic myocardium goes through a healing process, characteristic curves are obtained. By taking serial electrocardiograms the progress of the recovery process can be followed accurately and an evaluation of the patient's progress made which cannot be made in any other way. We can tell whether the myocardium is returning to a healed state or whether the recovery process is stopping before complete healing has occurred. In younger people the electrocardiograph tracing may return to a condition practically indistinguishable from that of a normal person. In older persons the recovery process usually goes forward and then becomes stabilized so that no further return to normal occurs. These latter usually have an abnormal electrocardiogram the rest of their lives. Repeated electrocardiographic tracings during the course of recovery from a coronary occlusion are of equal value in the management of treatment to the clinical findings.

I should like to discuss a few of the terms used in the interpretation of electrocardiograph tracings in order to answer a few of the most common questions that are asked regarding them. The interpretation of the electrocardiograph as made by most men in the field consist of a least three and sometimes four statements. First, the rhythm is described. Any sinus rhythm is usually considered normal. Then follows a statement as to the electrical axis of the heart. Until about two years ago this was stated as a deviation of so many degrees. At the present time fewer and fewer men are describing the axis deviation so closely, because Katz has shown that the Einthoven triangle, which supposedly projected the three dimensional qualities of the heart size onto a plane surface, is not accurate and that the factors upon which the electrical axis of the heart was determined are affected not only by the size of the heart but by its contact with the muscle masses surrounding it, the electrical conductivity of the tissues between it and the site of the leads used for making the electrocardiogram, and the position of the heart with relation to the chest cavity.[10] It is more accurate to say that the heart is either shifted toward, or is preponderant on, one side or the other. The third statement concerns the state of the myocardium. Many terms are used in this description: "active myocardial damage," "myocardial involvement," "myocardial degeneration" and "pathologic myocardium." They all probably mean the same thing and the terminology because of this is a matter of choice. I personally prefer the term "myocardial involvement" because "myocardial damage" is so frequently interpreted as meaning more than it should. The thought seems to flash through the mind of the reader of "myocardial damage" that it means a ruined heart. It does not necessarily mean this at all. The standards of normality for electrocardiographs are usually those of healthy young adults. Why should the heart of a fifty-year-old man be the same as that of a healthy young athlete of twenty? If our arteries and our prostates and our eyes and our skins change with age, why should not our hearts do the same? This statement as to the state of the myocardium must be interpreted by the clinician in the light of the clinical findings present.

I have tried briefly to outline some of the more important functions of the electrocardiograph. It may be that I have given the impression that it is the only important method of obtaining information about the heart. If so, I want to correct that impression. The electrocardiograph is a valuable instrument and in some instances gives information that can be secured in no other way. It has a definite place in our armamentarium. Levine has, I think, stated its case very well. He says: "Electrocardiography has become an essential part of our methods of exam-

ination of the heart. At present conditions can be recognized and treatment can be more intelligently directed which were entirely beyond the scope of the most experienced physician a generation ago. The information obtained by this new method must be appraised carefully and used as a part of a chain of data concerning the problem involved. At times a negative finding does not eliminate a suspected diagnosis and likewise a positive finding may have nothing to do with the major complaint. Normal electrocardiograms may be obtained in patients suffering from the most serious types of heart diseases, such as bacterial endocarditis and angina pectoris. Furthermore, when abnormal electrocardiographs are found, care must be exercised in interpreting them in terms of the patient's illness. An able clinician who knows nothing about the string galvanometer can still do better work than an expert in electrocardiography who has had limited experience and inadequate clinical judgment."[11]

We might compare our work to that of the photographer. If he is an expert in his profession and has had wide experience, he can produce fine pictures with simple equipment, but give this same man the best in equipment with all the necessary accessories and he will be able to produce excellent photographs under the most difficult conditions. Take away from the able clinician all of his laboratory aids and make him work with a stethoscope and his eyes and fingers, he will still produce a good diagnosis and adequate treatment. Give him all the accessories such as the x-ray, the clinical laboratory and the electrocardiograph, and he will, like the photographer, produce that perfect picture which we are all striving to create—an accurate diagnosis, prophetic prognosis and intelligent treatment.

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VARICOSE VEINS

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A varicose vein (varix, phlebectasia) is a permanent dilatation of one or more veins, usually with elongation, tortuosity, and thickening of the walls. The long and short saphenous veins of the leg are most often affected. It is a pathologic condition affecting a great number of people.

Many patients suffering from this condition do not consult their physician until complications arise, as the laity has not been educated to the fact that varicose veins may be simply and effectively treated if seen early.

EMBRYOLOGY

The embryology of the veins of the extremities as given by Prentiss and Arey[1] is as follows:

"The primitive capillary plexus of the upper and lower limb bud gives rise to a border vein, which courses about the periphery of the flattened limb buds. In the lower extremity the fibular portion of the primitive border vein persists. Later the long saphenous vein arises separately from the posterior cardinal, gives off the femoralis and posterior tibialis, and annexes the fibular border vein at the level of the knee. Distal to this junction the border vein persists as the anterior tibial and probably the small saphenous vein."

The embryology of the saphenous valves has been carefully studied by Kampmeier and Birch[2] on the human embryo, full-term fetus and venous system dissection in the adult cadaver. McPheeters[3] in his book reviews their work and the following is a superficial résumé of it.

They found no constant number of valves in any portion of the saphenous system. Kampmeier found constantly a valve 1½ to 2 centimeters above the saphenofemoral junction, and often two sets about 3 centimeters apart. Six to twelve valves were found, as a rule, in the long saphenous. There was an average of about four to six valve sets in the long saphenous above the knee. They found valves in both the long and short saphenous below the knee, but the number varied greatly. All the valves made their

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first appearance at three and a half months and seemed to have had their development by the fifth month of fetal life.

Valves were usually located at the entrance of the saphenous into the femoral and into the popliteal. They were found below the opening of a tributary; however, this was not constant and there seemed to be no regularity whatsoever

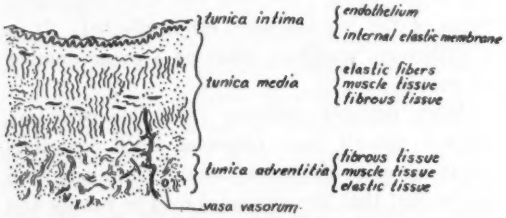

Fig. 1. Cross section of a vein.

in their location. They found the majority of valves in the saphenous system were of the bicuspid type as were also those of the other veins of the leg.

HISTOLOGY

The microscopic anatomy of normal veins is shown in Figure 1.

There is a layer of endothelial cells lining the inner wall of the tunica interna, with the elastic interna beneath. The next and largest layer is the tunica media with large amounts of muscle tissue interposed with fibrous tissue and scattered elastic fibers.

The tunica adventitia forms the outer layer of the vein wall. It is composed almost entirely of fibrous tissue and small amounts of muscle tissue running both longitudinally and circularly. The fibers of the elastic tissue run longitudinally, circularly and obliquely. The vasa vasorum course through the media and adventitia.

Anatomy

The venous system of the lower leg is composed of a deep and a superficial set of veins united by a rich network of anastomosis. In addition there are communicating veins connecting the two sets. These are variable in number and locations and are equipped with valves which permit the flow of blood from the superficial into the deep veins, but do not normally permit it to flow from the deep to the superficial veins. The valves in the saphenous veins prevent a reflux of blood and maintain the column of blood above them.

The deep veins composed of the femoral, popliteal, anterior and posterior tibial and their tributaries are located beneath the deep fascia and are surrounded by the powerful muscles of the leg, which prevent the veins from dilating when subjected to pressure. The pumping action of the muscles in walking aids in emptying the veins. Varicosities of the deep veins rarely occur for these reasons. The blood from the deep tissues about the foot and ankle collects in the deep veins which become the popliteal as it reaches the popliteal space and thence continues upward to Hunter's canal where it becomes the femoral until it passes under Poupart's ligament.

The superficial veins lie in the subcutaneous fat of the leg forming a network and consists of the long and short saphenous, and their tributaries. These veins lack the muscular support and pumping action of the muscles, which the deep set have, and therefore may dilate under excessive pressure and form varicosities.

The long saphenous originates from the foot and ankle medially, anteriorly, and courses upward until it enters the deep systems at the foramen ovalis.

The short saphenous originates from the foot and ankle, laterally and posteriorly, and passes upward until it joins the deep system in the popliteal space (fig. 2).

McPheeters[3] has found in his dissections that often along the entire course of the long saphenous there are small normal veins lying close to and often parallel with a varicose vein. He points out that this anatomical relationship may be the cause of a recurrence of veins a few years after injection treatment has been given.

ETIOLOGY

There is no general agreement as to the etiologic factor or factors in the production of varicose veins. Most investigators believe that a hereditary tendency supplanted by pressure is the most frequent cause. The subject is not settled and no satisfactory explanation can be presented in the majority of cases.

McPheeters[3] in his series of cases found the age limit to be from nine to seventy-seven years, with the highest percentage between thirty and forty.

Sex incidence, in McPheeters'[3] and Bernsten's[4] series, showed an equal distribution up to the twentieth year, after which women showed a

marked preponderance. More women present themselves for treatment, probably because of cosmetic reasons.

In reviewing the exhaustive amount of experimental work which has been done to determine the cause of varicose veins, it is safe to say that no one theory will explain all cases. Most investigators agree that there is a deficiency in the valves.

The *mechanical explanation* was given by Delbet.[5] He explained that the loss of valve function was the first step in all cases. The back pressure from the iliac vein, he believed, caused the valves at the saphenous femoral junction to give way, allowing a reverse flow to develop in the leg and the successive valves. Any act which contracts the abdominal muscles and increases the intra-abdominal pressure as lifting, coughing, sneezing, etc., or any cause of abdominal straining, as constipation, stricture of the urethra, enlarged prostate, etc., will produce back pressure. In a like manner any obstruction to the venous return, as pregnancy, pelvic tumors, thrombosis of femoral or lilac veins, cirrhosis of the liver, etc., will increase this pressure.

Valves do not lose their competency in succession. Klotz, quoted by McPheeters,[3] explains this from an embryonic standpoint. He believes there is a progressive degeneration of the valves with age, since it is known that many more valves exist during fetal life than are accounted for at birth and many do not survive the adult period.

The *endocrine factor* must be considered as of possible etiologic significance. Sicard's[6] theory is that there are three ovarian or endocrine periods in a woman's life, namely: before puberty, from puberty to the age of menopause, and during menopause and thereafter. He correlates the fact that bluish cords often appear at puberty, increase with each menstruation period, until pregnancy, when they increase in size, and finally at the menopause become quite large. This is often noted clinically. This theory, however, lacks proof. Although the endocrines are supposed to be at fault, the administration of extracts of these glands apparently does not benefit the varicosities.

The *hereditary* factor must be considered as playing a part. This is manifested by general connective tissue weakness, loss of nerve and muscle tone in the walls of the veins, and congenital weakness of the valves. de Takats and Quint[7] elicited a hereditary factor of 65 per cent. in their series of cases. Dawkins[8] states that sometimes the same segment of the same vein is the only one affected in members of the same family. There is a question as to whether the primary factor is an inherent weakness and deficiency of the valves, or an inherent weakness of the vein wall itself. Many people doubtless have a mild degree of inherent valvular weakness, but never develop varicosities because no exciting factor enters their lives for a sufficient period to exert a deleterious effect.

The *infection theory* is supported by some since varices are often a sequel of typhoid fever, diphtheria, pelvic infections, etc. It is believed that the infection attacks the vein wall either by direct extension from a nearby focus or from embolic or hematogenous origin. The infection produces a phlebitis or periphlebitis which may be of such a low grade type that no symptoms are noticed, yet the wall of the vein is weakened Primary implantations on the valves and vein wall have been cited as a cause. The blood current is most stagnant above the normally functioning valves. Should the blood stream be carrying an active infection the valves and vein wall would be attacked. A replacement of the elastic and muscular tissue by fibrous tissue would result and there would be a destruction of the valves with the development of varices.

Occupation and our erect position may be contributing causes. The main factors responsible for the venous return of the blood from the lower extremities are: 1. *Abdomino-thoracic suction;* 2. *skeletal muscular contraction and relaxation.* and 3. the *vis a tergo,* which Dawkins[8] describes as the force with which the blood enters the venous radicles from the capillaries. It is a continuous acting force and during muscular relaxation forces blood into the small veins, past the first valves when it comes under the influence of muscular contraction. Therefore, in those whose occupations demand long hours of standing, as barbers, clerks, etc., this pumpng action is lost and continuous back pressure of the venous column results. The saphenous is the longest vein in the body and therefore is the one most commonly affected.

The *vitamin theory* is the most recent. It is

based upon the absence of vitamin C. This vitamin seems to have a definite and important relation to disorders and disease involving the endothelial structures and blood vessel walls. McPheeters[3] states that research workers have produced evidence that its absence may have a direct bearing upon the development of varicose veins. He thinks it is still open to question but worthy of thought and trial therapy.

CLASSIFICATION

The classification of varicose veins according to the pathologic formations present or the types of varices as given by Bernsten[4] is most serviceable. The following classification is taken from McPheeters' book:[3]

Type 1 is the isolated saccular varix, usually appearing along the course of the great saphenous veins.

Type 2 is the fully developed, tortuous, cavernous mass of varices, either in the great or small saphenous vein.

Type 3 is the varix of uniform dilatation of a large trunk. The walls of these veins are markedly hypertrophied and undergo a dilatation with persisting increase in pressure. This may be seen on all parts of the thigh and lower leg.

Type 4 consists of five cutaneous dilatations of a stellate design and are similar to what McPheeters calls "skyrocket" or "spider bursts." They are usually seen on the external surface of the thigh in fleshy women.

The size and diameter of varicose veins as well as their type and location are essential points in any discussion. McPheeters[3] is probably the first to establish such a gradation. His grouping is as follows:

Size 1: Veins ½ cm. in diameter.

Size 2: Veins ½ to 1 cm. in diameter.

Size 3: Veins 1 to 1½ cm. in diameter.

Size 4: Veins 1½ to 2 cm. in diameter.

Size 5: Varices above Size 4 are in reality no longer "veins" but are mere saccules of blood and need a definite description as to their size and shape.

PATHOLOGY

Although the cause of varicosities is not definitely known, we do understand the tissue changes which take place. The collagenous tissue of the intima proliferates and invades the muscular and elastic tissues. Round cells are found around the nutrient vessels and secondary calcification may take place. The veins lose their elasticity and become very thin, the valves become incompetent, and the pressure in the vein increases. This increased pressure along with the alterations in the veins allows blood to escape into the tissues with the result that edema, loss of tone, pigmentation, poor nutrition, inflammations, dry scaly or eczematous skin and ulceration may occur.

The stagnation of blood from increased pressure in varicose veins must result in changes in the blood chemistry. de Takats et al[9] have shown in carefully controlled experiments that the carbon dioxide content of the varicose blood is definitely higher than that of the venous blood in the cubital veins of the same person. The reverse is true of the oxygen content. Blalock,[10] in his experiments, found that when ulceration was not present the venous oxygen content of varicose veins of the lower part of one extremity is lower than that of similarly placed veins of the opposite extremity. However, when ulcerations and infections were superimposed on the varicose veins, the oxygen content was usually higher on the diseased side.

It has always been the assumption that in varicose veins the upward flow of blood is markedly slowed or reversed and that varices remain as dilated tubes of blood. McPheeters[3] states that the Trendelenburg test is merely a test proving the reverse flow of blood in the great saphenous veins from the saphenofemoral junction downward. This test is positive in all cases of extensive varicose vein development. He further states that this same phenomenon of a downward or reverse flow develops from a "blow out" or wide dilatation of any of the communicating veins from the deep system any place along its course upward, but it occurs most often in the lower leg as the communicating branches are more apt to dilate in this location.

McPheeters,[11] by injecting lipiodol high into the saphenous veins of a varicosed leg and observing it under the fluoroscope, demonstrated that the lipiodol traveled downward or reversely and part of it gained access to the deep circulation, during exercise, by way of the communicating veins. None was seen to go up through the superficial saphenous veins. This phenomenon, he believes, explains why emboli rarely develop

following injection treatment. McPheeters[12] further proved by blood pressure experiments that the blood is slowed or stagnant in any case of varicose veins. When the patient is walking the flow actually may be reversed, being outward from the femoral via the communicating and downward through the varicosed saphenous system. Schmier's[13] results, however, when he injected iodized oil into the superficial system in two cases, did not coincide with the more extensive work of McPheeters. He concluded that the complete reversal of the venous system was not as common as was ordinarily supposed.

The pathology of thrombus formation in varicose veins treated by injection is described by Lufkin and McPheeters.[14] They found thrombus formation depended upon an injury to the endothelial lining of the vessel. The total occluding mass is composed of (1) a white accumulation (Welsh) or deposition (Aschoff) thrombus, which due to the extensive intimal damage provides adequate attachment for the thrombus, and is the smaller mass, and (2) a red, coagulation thrombus, or simple clot, which makes up the larger mass. They believe recurrences probably result from retraction of this clotted or red portion of the mass from the vessel wall. Theis[15] states that this coagulation or secondary stagnation thrombus is particularly prone to cause emboli because of lack of intimal attachment.

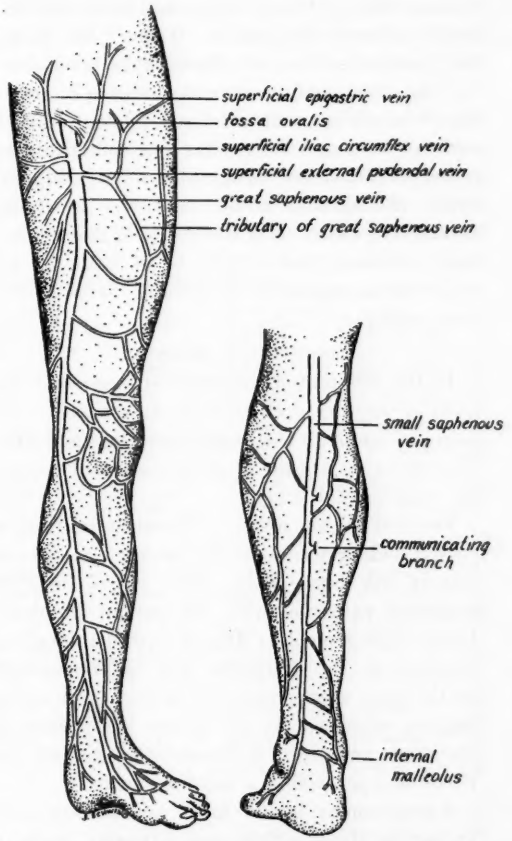

Fig. 2. Superficial venous system of the lower extremity.

SYMPTOMS

Varicose veins may cause relatively few or no symptoms. Faxon[16] believes that any symptoms at or above the knee are, with few exceptions, not due to varicosities. Ochsner[17] states that the symptoms of varicosities are not proportionate to the dilatation. Some patients with small veins may have marked symptoms, while others with markedly dilated veins may have none.

The symptoms most frequently complained of are heaviness and a tired feeling in the extremities, especially after standing. Fatigue is a frequent symptom. Pain and aching which at times may resemble a cramp is occasionally complained of. Burning, itching, tingling and edema are other symptoms. Logefeil[18] states that 26 per cent. of the cases treated by him were for cosmetic reasons only, no symptoms being elicited.

DIAGNOSIS

The diagnosis of varicose veins is usually not difficult, but hidden varicosities due to excessive or brawny edema may occasionally cause confusion. The physical findings in varicosities consist of varying degrees of dilatation and tortuosity of the veins. The large dilatations, as a rule, are located on the posterior or medial aspect of the knee. In very obese individuals the varicosities cannot be easily visualized. Transmission of an impulse along the course of a vein is indicative of varicosities and also incompetency of the venous valves.

The diagnosis of varicose ulcer may be more difficult. Schmier[19] states that diabetes, trophic distrubances, syphilis, trauma, chemical irritation, phlebitis and postoperative complications are often associated with varicose ulcers.

Infra-red photography has been used by Barber[20] and Zimmerman[21] to visualize superficial

veins, visible concealed varicosities, and venous dilatations due to obstruction. Wilson[22] has shown the changes in infra-red photographs taken during the treatment of varicose veins. Zimmerman[23] concludes that infra-red photographs of varicose veins and their cutaneous complications reveal an immediate topographic relationship between the dilated trunks and the overlying cutaneous lesions. McPheeters[3] is of the opinion that infra-red photography is of little or no value in the treatment of varicose veins of the lower extremity.

DIFFERENTIAL DIAGNOSIS

In the differential diagnosis of varicose veins must be considered all those diseases which cause pain or cramps in the knees, lower legs and feet. The following are the most frequent and require the most careful study.

Femoral hernia must be dfferentiated from a circoid enlargement of the internal saphenous vein in the fossae ovals. This can be done by bimanual palpation with the patient standing. If the enlargement is due to circoid dilatation, pressure on the dilatation will be transmitted in the long saphenous vein in the form of an impulse which can be felt in the lower third of the thigh and leg. McPheeters[24] calls this the P. P. T. (percussion pulse transmitted).

Arteriovenous fistula must be differentiated. It can be diagnosed if one extremity is more moist and warmer than the corresponding side or if there is evidence of a thrill or bruit. Further evidence can be obtained if the measurement from the anterior superior spine of the ilium to the lower tip of the internal malleolus on the same side shows a lengthening as compared to the other side. Determination of the oxygen content of the blood plasma will also help to make a differentiation.

Rheumatism of the knee or ankle as well as arthritis of the hip, knee or ankle must be considered in the differential diagnosis. Arthritis occurring in fleshy women during the menopause may often cause varicose veins and the pain is attributed by the patient to the veins. McPheeters[3] advises injecting the veins as he believes they aggravate the arthritic symptoms.

True sciatica usually travels along the posterior thigh and calf to the ankle. Neuritis, polyneuritis and tabetic pains may complicate the picture. Blood Kahn in the latter will usually make the differentiation.

Osteitis and periostitis are generally associated with a previous history of trauma.

Single or multiple herniations of the muscle sheath, as discussed by Smith,[25] may stimulate varicose veins. Both conditions may be present simultaneously.

Spurs on the os calcis will many times cause pain upon standing, and flat feet cause pains and aches in the calves of the legs.

Figure 3 is a composite chart of the differential diagnosis of the vascular diseases affecting the extremities as compiled by McPheeters[3] and is presented here with the permission of the author and the publisher.

TREATMENT

The treatment of varicose veins resolves itself into three classes:

1. Palliative.
2. Injection of sclerosing agents.
3. Injection plus ligation of the saphenous veins.

The older methods of operative treatment as advocated by Trendelenburg, Schede, Pierre Delbet, Rindsleisch, M e d e l u n d, Patzenstein, Schwartz, C. H. Mayo, Babcock and others have practically been abandoned. On the other hand, Homans[26] as late as 1928 was skeptical of the injection treatment and advised operative procedure.

1. *Palliative Treatment.*—This consists in the wearing of elastic stockings or bandages, or in the application of elastoplast or tensoplast bandages. The latter contains an adhesive substance which exerts a better and more even pressure and keeps the bandage from slipping. Such a bandage should be removed after two to four weeks and a fresh one applied. Elastoplast or tensoplast bandages merely give support to the dilated varices mechanically, holding their walls collapsed or compressed to their normal size. Palliative treatment is often indicated in pregnancy, since after the termination of labor the veins may regain their normal tone and bandaging will no longer be necessary.

2. *Injection of Sclerosing Solutions* into the blood vessels with the object of producing thrombosis was first employed in 1851 by Pravaz who was the inventor of the hypodermic syringe. He attempted to cure aneurysm by the injection of ferric chloride solution. In the succeeding years

many European investigators tried various solutions but little progress seems to have been made until 1911, when Professor Linser of Tübingen, Germany, observed that following injection of mercuric chloride intravenously for syphilis the veins of the arm became obliterated after many injections. In 1916-17, Professor Sicard of Paris observed the same phenomenon taking place in soldiers treated during the war with intravenous Sodium morrhuate in 1 or 2 cc. doses has been most widely used. Very few systemic allergic reactions were encountered, although they have been reported by several writers as Glick,[27] Lewis,[28] McCastor,[29] Dale,[30] and Traub.[31] McPheeters[32] believes these reactions occur when the patients have been overtreated by injecting too large a quantity of solution into one varix of the calf. He advocates the use of a tourniquet

DIFFERENTIAL DIAGNOSIS OF VASCULAR DISEASE AFFECTING THE EXTREMITIES*

	Varicose Veins	Arteriovenous Aneurysm	Thrombo-angiitis Obliterans Buerger's Disease	Arteriosclerosis	Raynaud's Disease and Similar Conditions	Primary Erythromelalgia
Age	From puberty on	Present at birth	Mostly between 25 and 40 years	Mostly between 55 and 85 years	Mostly between 17 and 35 years	Mostly between 35 and 50 years
Sex	Females 54%	Equal	Males 99%	Males 90%	Females 95%	Both sexes 10%
Nationality	Any	Any	Hebrews 50%	Any	Any	Any
Heredity	Dominant influence	Congenital	Not known	Marked influence	Marked influence	Not known
Pulsation of arteries	Normal	Normal	Pulseless 50% Diminished 45% Normal 5%	Pulseless 50% Diminshed 45% Normal 5%	Normal	Normal
Dependent rubor	Absent	Absent	Present	Present	Absent	Absent
Pallor on elevation	Absent	Absent	Present	Present	Absent	Absent
Claudication	Absent	Absent	Usually present	Usually present	Absent	Absent
Rest—pain	Usually absent	Absent	Severe, sharp and stinging	Mild aching	Usually absent	Burning
Cramps and tingling on standing	Frequent	Frequent	Mild	Mild	Usually absent	Absent
Ulcers	Moist about ankles	Seldom	Inflamed, discharging on toes	Usually dry above prominences	Small punched out areas	None
Phlebitis	Frequent	Seldom	30% of cases returning	Absent	Absent	Absent
Edema	Frequent	Absent	Frequent	Infrequent	Absent	Absent
Roentgenogram of arteries	Negative	Negative	Usually negative	Positive for sclerosis	Negative	Negative
Color changes on exposure	Normal	Normal	30%	15 to 20%	Always	Never
Skin temperature	Normal	Increased over arteriovenous anastomosis	Low	Low	Low	High
Histamine reaction	Normal	Normal	Absent	Absent	Normal	Not tested
Response to foreign proteins	Marked	Marked	Marked	Slight	Marked	Marked
Oxygen content, venous blood	54	90	Normal	Normal	Normal	Normal

* Combined statistics of Allen, Brown and Mahorner of the Mayo Clinic, DeTakats and Quint of Northwestern University, and the Author.

Fig. 3.

Para Courtesy of McPheeters and Anderson, and the F. A. Davis Company.

luargol. He soon recognized that the sclerosing and resulting obliteration was due to the free soda in the solution. In 1918, he found that neutral sodium carbonate led to thrombosis and sclerosis of the veins, but because of the extensive sloughing which it produced in the subcutaneous tissues it was soon abandoned in favor of the much less toxic sodium salicylate.

The author in the past twelve years while working in the Mercy Free Dispensary and in private practice, has used sodium salicylate, invert sugar, quinine and methane, varisol, sodium morrhuate, and more recently monolate (monoethanolamine oleate, 5 per cent.). All except the latter two have practically been discarded. in an effort to localize the injected solution, thereby retarding its rate of absorption and minimizing the allergic response.

Occasionally a varying degree of shock is encountered, as described by Smith.[25] This may vary from a fainting spell to surgical shock which is manifested by a total collapse, ashen color, disappearance of the pulse, loss of reflexes, cold, clammy skin, dilatation of the pupils, and sweating. The writer has noted attacks of syncope on two occasions in the same patient, after the use of sodium morrhuate in the first injection and monolate in the second injection. A psychic reaction is probably the explanation of this and many other instances.

TECHNIC OF INJECTION

In his work the writer has adopted the following technic. The patient stands either on the floor or a low chair. A rubber constrictor is applied usually above the knee. The site of injection is determined and carefully cleansed with alcohol and iodine. A 23-gauge, short beveled needle about one and one-half inches long is used, attached to either a 2 or 5 cc. Yale slip-on or Luer lock control syringe, depending upon the size of the vein and the amount of solution to be injected. Great care is exercised to place the needle in the lumen of the vein and not through it. When the blood is aspirated back into the syringe freely, the solution is slowly injected. The patient should experience no pain during the injection if the needle is properly inserted into the lumen. The needle is quickly withdrawn and pressure applied for a few seconds with the finger over a cotton sponge to prevent the solution from leaking out into the perivascular tissues. A piece of gauze, ¼x½ inch in thickness, is then applied over this area and held securely in place by a piece of adhesive tape. The tournique is removed. In most instances the patient is instructed to return in one week. Many times, however, when there are numerous large dilated veins an elastic support or ace bandage is wrapped around the leg from the foot to just below the knee. The patient is instructed to remove this bandage at night and reapply each morning until returning for the next treatment. A cramping sensation in the lower leg is occasionally complained of by the patient due to a spasm of the venous walls but this usually disappears if the patient is instructed to elevate the leg upon a chair for a few minutes.

INDICATIONS AND CONTRAINDICATIONS

A thorough knowledge of the indications and contraindications for injection treatment is quite imperative. The indications vary with each individual clinician and with each patient. Most investigators agree that (1) varices which are so large and painful as to incapacitate the patient, and (2) varices complicated by eczema, pruritus and ulcer should be injected provided there are no contraindications. McPheeters[3] believes that all varicose veins should be injected provided there are no definite positive contraindications. He further believes that cosmetic reasons which include flairs and bursts are sufficient indication provided the patient desires it.

The contraindications to injection treatment are listed as follows:

1. Incompetent or closed deep venous system in which the superficial varicosities are compensatory. This can be determined by the Trendelenburg and Perthes test, which will be described, or by the combined tourniquet test described by Mahoner and Ochsner.[33]

In the Trendelenburg test the patient lies down upon a table and the foot and leg are elevated high. The blood from the varices is thereby drained into the general venous system by the force of gravity. A tourniquet is applied tightly about the upper or middle thigh and the patient instructed to stand up. In any well developed case of varicose veins those veins above the tourniquet will fill up rapidly while those below will remain collapsed. If, when the tourniquet is suddenly removed, the lower varices fill rapidly the conclusion is drawn that they have filled with the blood from above. This phenomenon is termed a *Trendelenburg positive*. When the test is made with the tourniquet tightly applied and the patient standing, if the varices fill quickly from below, then it is clear that the varices have filled from the deep system due to the incompetency of the valves in the communicating veins. This is called a *Trendelenburg negative*.

In some cases the varices will fill rapidly from below but when the tourniquet is removed they will fill more tensely. This shows that the blood has come outward from the deep system through the communicating veins into the superficial system, and also downward through the great saphenous from the faulty valves above. This is called a *Trendelenberg double*.

The veins of the lower leg and thigh occasionally become distended, yet all the valves are competent. The varices in this case would simply fill slowly with the blood returning from the distal parts of the extremity. This is called a *Trendelenburg nil*.

The Perthes test is performed in the following manner: A constrictor is applied about the thigh with just enough pressure to shut off the superficial circulation while the patient is standing, and the superficial veins are engorged. The patient then walks about for a few minutes. Due to the pumping effect of the alternate muscular con-

tractions and relaxation, the return of blood through the deep veins is facilitated and if these are patent the vein will empty or decrease in size during activity with the tourniquet applied. A variation of this test is to determine whether the veins will empty by gravity when the limb is elevated, while the constrictor is still in place. Since such emptying cannot take place through the superficial veins, it must indicate that the deep veins are patent, and the superficial veins may be injected with safety.

2. Obstruction of the venous return of the leg by pelvic or abdominal tumors. If the varices persist after the tumor has been removed surgically they may be safely injected.

3. Recent thrombophlebitis of the deep system is a positive and definite contraindication.

4. A superficial phlebitis, acute or subacute, contradicts the injection treatment. Six months or longer, according to some investigators, should elapse before attempting treatment. de Takats[34] believes the trauma of injection may reactivate the resting infection in the veins.

5. In pregnancy after the seventh month injection is contraindicated. Most investigators agree that injections may be safely given up to the seventh month if indications are present, but not after. The author has always treated these patients palliatively, never injecting until some time after the termination of labor if the veins persist and give symptoms.

6. Bedridden patients, especially in the older age group, should not be given injection treatment. Patients receiving injections should be ambulatory. The only fatal case of pulmonary embolism in the writer's experience occurred in an elderly lady who presented herself at the clinic with varicose veins and a varicose ulcer. She was given the usual injection. Upon returning to the clinic later she decided to enter the hospital for treatment of the ulcer. She was again injected. She remained in bed for almost two weeks, by which time the ulcer was almost completely healed. Suddenly during the night on the eve of her departure she expired. Autopsy findings revealed a long pulmonary thrombus which had migrated from the injected leg.

7. Buerger's disease (thrombo-angiitis obliterans) according to Kilborne[35] is an absolute contraindication to the injection treatment. He cites a case in which bilateral amputation of the leg was necessitated due to gangrene developing following infection.

Fig. 4. Syphilitic ulcer associated with varicose ulcer.

8. Raynaud's disease, advanced senile arteriosclerosis and similar conditions of the circulatory system are contraindications.

9. Portal blockage from cirrhosis of the liver or other obstruction.

10. Diabetes mellitus, if not under proper management and if not controlled. In the earlier days when sugar solutions were being used, diabetes was an absolute contraindication, but not necessarily so with the more recent solutions.

11. Other contraindications are severe nephritis, malignancy, severe cardiac disease, including coronary disease, pulmonary tuberculosis, hyperthyroidism, subacute pelvic infection, gen-

eral debility in elderly patients, elephantiasis, and during the progress of a cold, acute tonsillitis or pharyngitis.

3. *Ligation of the great saphenous vein plus* injection is indicated in varicose veins of Size 3 and larger where there is a concomitant dilatation of the great saphenous vein. Injections alone fail in these cases due to the recanalization of the sclerosed segments and to the dilation of previously existing normal collaterals. Edwards[36] gives the history of saphenous ligation showing the upward progression of the elective site of operation, beginning with Celsus in the first century. Various investigators through the following centuries ligated the vein, each progressing upward until Homans in 1916 insisted that the ideal operation for varicose veins was to ligate the saphenous and all its terminal branches at the saphenofemoral junction. He surgically removed at that time all the varices below. The injection of the distal part of the vein with sclerosing solution as is done today serves the same purpose. de Takats,[37] in 1930, described the ambulatory ligation of the saphenous vein in patients with the positive Trendelenberg. He believed that the operation not only reduced venous pressure but also served as a barrier to ascending thrombi which might be caused by later injections. Sarma[38] in 1,000 ligations concluded that satisfactory results were obtained in Trendelenburg positive cases where the saphenous vein was ligated followed by injection of sclerosing solution until all the veins were obliterated. He found the most unsatisfactory results in the Trendelenburg double group of cases and states that the Trendelenburg negative group required no ligation, only obliteration by injection. The ideal site for ligation is as near the saphenofemoral junction as possible. When the saphenous is ligated distally there is always the possibility of a thrombus forming in the proximal segment and the potential danger of this thrombus entering the general circulation causing a fatal pulmonary embolism. McPheeters[32] states that he has never seen a case that might point to a pulmonary embolus in the combined method of treatment.

This combined method of high ligation plus injection appears to be the best treatment, at the present time, of extensive varicose veins with a marked reverse flow. This is exemplified by the work of McPheeters,[32] Edwards,[36] Swinton,[39] Faxon,[16] Hawkes,[40] Zimmerman,[41] Ochsner,[42] Haggard,[43] and others.

The writer's usual procedure is to hospitalize the patient for 24 hours, during which time the vein is doubly ligated under local anesthetic in the operating room, severed, and a segment removed. The distal end is usually injected with 2 to 5 cc. of sodium morrhuate. The patient is kept ambulatory and further injections given, if necessary, at weekly intervals. He has never done a bilateral ligation as one operative procedure.

COMPLICATIONS

Varicose ulcers are the most frequent complications of untreated varicose veins. They are usually located on the anteromesial surface, middle and lower thirds, over the tibia. Injection of the vein feeding the ulcer or injection plus ligation of the long saphenous vein with the necessary supportive bandages and sponges over the ulcer usually is sufficient to cure the ulcer.

Syphilitic ulcer may be associated with varicose ulcer (fig. 4). It is usually located upon the anterolateral surface of the knee. The syphilis should be treated simultaneously with the treatment of the ulcer.

Tuberculous ulcers (Bazin's disease) are usually located in the anterolateral surface of the upper and middle third of the lower leg. Supportive and local treatments are of value in the management of these ulcers.

Hemorrhage from a ruptured varicose vein has occasionally been encountered. Elevation of the leg and the application of pressure over the ruptured veins will usually suffice, but occasionally the writer has found it necessary to ligate the veins above and below the bleeding point under local anesthetic.

Chronic eczematous areas of the lower leg may complicate varicose veins. Occluding the varicose vein and applying an Unna's boot or elastoplast bandage is practiced by some, while others prefer to use the supportive treatment, elevation of the leg and the application of antipruritic ointment until the case is under control, and then ligate and inject as in any other case. McPheeters[32] is in favor of this supportive treatment.

Complications following the injection treatment are of utmost importance. Ulceration at

the site of injection does not occur as frequently as formerly when the more irritating solutions were used. The injection of normal saline or distilled water in the area is of value in neutralizing the caustic effect and thereby may prevent ulceration. McKinstry[44] states that if ulceration results radical excision should be done as soon as the extent of the damage is determined. The technic of the operator will in many cases determine the frequency of this complication; however, in a large series of cases it will probably occur at some time or other.

Embolism has been the most feared of complications of the injection treatment. Fortunately it is rare and not all emboli are fatal. Kilbourne,[45] in 1934, reported his experience covering 20,000 injections without a mortality. He studied 20 deaths following injection treatment of varicose veins occurring in other clinics. The major portion of the deaths was due to bacteremia, bacterial phlebitis and embolism following bacterial phlebitis. Since emboli are usually due to infection the prevention of infection will reduce their incidence. The reverse flow of blood in varicose veins is an aid in preventing the migration of emboli. Keeping the patient ambulatory is a further safeguard against embolism. This has previously been emphasized in this paper where the fatal embolus occurred in the elderly bedridden patient. Infection from the previous ulcer probably helped to predispose to the emboli formation.

Infectious thrombophlebitis is a common complication of varicose veins and, according to Edwards,[46] occurs at some time in the course of about one-half of all patients with varicose veins. He believes the saphenous veins should be ligated at the saphenofemoral junction and followed by regular exercise and if necessary by application of heat. He further states that small injections of sclerosing solution may be used if some of the varices are still open. He reports 63 cases treated in this way with good results. The majority of investigators, however, treat this condition conservatively. The writer's plan has been to employ conservative treatment and not to inject.

Phlebitis may occur during the course of treatment. The infection rarely reaches the deeper vessels. Occasionally the reaction is out of all proportion to the amount of solution injected. This is probably due to the stirring up of a latent or resting infection as described by de Takats.[34] There is swelling with redness and considerable discomfort for about a week. The infection then subsides, leaving a well thrombosed vein. Injection is contraindicated for several weeks or months when this complication arises.

Allergic reactions are more frequent following sodium morrhuate and quinine injections. As previously stated, this complication has not been frequent in the writer's experience.

Pulmonary infarction, as described by Smith[25] has not been observed or has not been recognized in the writer's experience.

Pain in the back, similar to lumbago, has been noted occasionally especially when the stronger solutions were used. Smith[25] explains this as being due to a stimulation of the sensory branch of the lumbosacral plexus, as it emerges from the deep tissues on the medial surface of the knee.

SUMMARY

1. A study of varicose veins, covering embryology, histology, anatomy, etiology, pathology, diagnosis, differential diagnosis and treatment is presented.

2. The technic of the injection treatment is described.

3. Indications and contraindications for injection treatment are given.

4. Complications are emphasized.

5. A case of fatal pulmonary embolism is cited.

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THE USE OF BEE VENOM IN THE TREATMENT OF NEURITIS AND ARTHRITIS

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The use of venom in the control of pain was apparently known to the ancients, it being mentioned by Hippocrates[3] in his writings. The belief that bee venon was useful in cases of neuritis, muscular pains, sciatica, and various forms of arthritis came from the observation that bee keepers were seldom afflicted with rheumatism. There were also rumors and reports that persons having rheumatism who accidentally were exposed to numerous bee stings reported a complete relief from pain and increased mobility in the stiffened joints.

The early workers studying bee venom therapy used the live bees. They controlled the number of stings by collecting the bees in a glass bottle. They then transferred the number of bees desired to a cupping glass inverted over a piece of paper. The glass was then transferred to the skin of the person being treated and the paper slipped out. The bees began to sting immediately. As many as thirty bees were permitted to sting the patient at one sitting and this was repeated every three or four days for as long as two months. Perrin,[3] in 1933, produced an injectable form of bee venom by removing the venom sacs with flamed forceps, extracting

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the venom in absolute alcohol, then drying in a vacuum. The resultant powder was then dissolved in normal saline and injected hypodermically.

The exact chemical composition of bee venom is not known. Burt[13] states that been venom is a combination of an albumin-free sapotoxin allied to snake venom and a poison similar to cantharides, and that it is quite free from formic acid. It has been a prevalent belief for centuries that formic acid constituted the potent part of the venom. The venom is easily destroyed by alcohol, iodine and by oxidation. One suggested method of standardization was to inoculate the venom in the tail of a mouse. One bee sting would be the amount to kill the animal.

There is considerable controversy as to the action of venom in the relief of pain. Some think it is due to protein shock, others consider it a desensitizing action and others think it is simply a counterirritant, producing a capillary engorgement at the site of inoculation. The production of histamine was also considered. Burt,[13] however, showed that the injection of histamine did not produce a similar reaction to that of bee venom when injected into sensitized patients. Starkenstein and Weden[9] in 1936 demonstrated that the action of saponin is due largely to a disturbance in the distribution of cholesterol. This disturbance of distribution causes a change in the permeability of the cellular walls of the different organs, thereby causing changes in the distribution of exogenic and endogenic substances. Beck[6] states that there are two physiological effects of bee venom. First, hemorrhagin, which stimulates circulation, renders the capillaries more permeable, thus carrying more oxygen to the tissues, increasing metabolism, supplying heat, and improving elimination. Second, the neurotoxic effect by paralysis of the nerve endings of the sympathetic nervous system, releases the capillary constriction, and in this manner increases the blood supply to the affected part. The venom also produces a paralysis of the sensory nerve endings and it is thought that its pain-alleviating effect is due to this property.

The use of bee venom is mainly restricted to the relief of pain in the rheumatic conditions such as the various forms of arthritis, myalgias, neuritis, sciatica, etc. The more potent snake venoms have been used successfully in the relief of the more intractable pain associated with malignancies and in the control of hemorrhage associated with hemophilia, functional uterine bleeding, purpura, epistaxis, Osler's disease and in hemoptysis from bronchiectatic cavities.[11] The use of cobra venom in arthritis has not been very successful, however, there has apparently been no extensive scientific study with reference to this matter.[12]

The technique as used in this series of cases, and as given by the manufacturer of the bee venom solution, is as follows: The injections should be made intradermally and as near the seat of pain as possible, except that it should not be injected about the neck and face. The injections are made by means of a tuberculin syringe and a small 26 or 27-gauge short beveled needle. The skin should be cleansed with ether since alcohol precipitates the venon. The first injection should be small, 0.01 to 0.02 cc. or 1/10 to 2/10 of one bee sting. Each succeeding dose, given at two- or three-day intervals, should be increased by 0.01 to 0.02 cc., depending on the reaction obtained, until each injection is equivalent to 0.1 cc. of one bee sting. When the total amount of one injection equals one bee sting or 1/10 of one cc., each subsequent dose can be increased by one bee sting or .1 cc., using multiple injections about ½ inch apart of .1 cc. each. The number of injections can be increased at two- to three-day intervals until a total of ten bee stings are given at one time or until a secondary reaction is obtained.

The type of reactions associated with the use of bee venom have been classified as primary, secondary and generalized. The primary reaction consists of the broad flat wheal obtained by the intradermal injection, surrounded in a few minutes by a diffuse deep red blush. If the secondary reaction occurs there will be a more extensive urticarial wheal develop which may show pseudopodia and considerable edematous swelling, accompanied by burning and itching. The general reactions in patients who are extremely sensitive to bee venom may consist of headache, vertigo, diuresis, diarrhea or generalized urticaria. In the true rheumatic individual the primary reaction is usually all that is noticed during the first few injections. In the non-rheumatic the secondary type of reaction is

usually seen and it seems that if the secondary type of reaction occurs during the first few doses, the patient is not apt to benefit much by venom therapy. The variation in reactions is probably due to the fact that arthritics seem to have a much higher immunity to bee venom than do normal individuals.

This series is composed of twenty-two unselected cases, in which there are eight males and fourteen females ranging in age from twenty-six the eighty-three years. Complete physical examination was done in each case to determine, in so far as possible, the etiology. There was only one case in the group with a rapid sedimentation rate; an atrophic or rheumatoid arthritis involving the finger joints and the right shoulder. There were nine case in which there was a definite focus of infection such as teeth, tonsils, gall-gladder, etc. Insofar as possible these foci were removed before or during the treatment. There were seven in the group with a neuritis associated with a hypertension and arteriosclerosis. In all of this group the pain involved the back of the head and neck, running down to the shoulders. There was also an associated hypertrophic arthritis in three of these patients. Their chief complaints were not with reference to the arthritis, but to the severe pain in the back of the head and neck. Treatment of the hypertension with subsequent reduction in the systolic pressure did not relieve the pain, but, if there was a significant reduction in the diastolic pressure, there was some relief from pain. All of this would lead us to believe that an increased blood supply to the affected parts plays a significant role in the relief of pain.

There were six cases in this group which had a neuritis associated with the menopause, three of whom had definite foci of infection in the teeth. One patient thirty-eight years old had a severe neuritis of pregnancy, so called because no other etiological factor could be found. The neuritis involved the right arm and hand and was so severe that it caused considerable swelling and disability. The use of bee venom in this case was entirely unsuccessful, as was other forms of therapy including the vitamins.

The classification used is that of Ainlay,[17] first as to whether acute or chronic, second as to etiology and type of involvement, and third as to location. There were 14 or 63.7 per cent. chronic and eight or 36.3 per cent. acute cases. Trauma was apparently responsible for two, infection nine, and 11 were of undetermined etiology. As to the type of involvement 12 were classified as neuritis, three as hypertrophic arthritis, four as sciatica, which of course is a neuritis, two as muscular and one as atrophic or rheumatoid arthritis.

TABLE No. 1

Type	Cases	Per Cent
Acute	8	36.3
Chronic	14	63.7

Etiology

Type	Cases	Per Cent	Acute	Chronic
Traumatic	2	10	1	1
Infection	9	40	5	4
Undetermined	11	50	3	8

Classification

Type	Cases	Per Cent	Acute	Chronic
Neuritis	16	72.8	8	8
Hypertrophic arthritis	3	13.6	0	3
Muscular	2	9.1	0	2
Atrophic arthritis	1	4.5	0	1

Table No. 2 lists the location and classification of each individual case. It will be noticed that 72 per cent. of the cases listed were classified as neuritis. Some of these were difficult to classify as to whether they were truly a neuritis or a myalgia, however, they were definitely extra-articular in character.

TABLE No. 2
Location and Classification

	Age	Sex	Class	Stage	Etiology
			Head and Neck		
1.	62	Male	Neuritis	Chronic	Undetermined
2.	50	Male	Neuritis	Acute	Undetermined
3.	50	Female	Neuritis	Chronic	Undetermined
4.	69	Female	Neuritis	Chronic	Undetermined
			Shoulders and Arms		
5.	39	Female	Neurities	Chronic	Infected
6.	56	Male	Hypertrophic Arthritis	Chronic	Undetermined
7.	33	Female	Neuritis	Chronic	Infected
8.	62	Female	Neuritis	Chronic	Undetermined
9.	52	Female	Neuritis and Sciatica	Acute	Infected
10.	40	Female	Atrophic Arthritis	Chronic	Infected
11.	42	Female	Neuritis	Acute	Infected
12.	62	Female	Muscular	Chronic	Undetermined
13.	38	Female	Neuritis	Acute	Undetermined
			Chest and Back		
14.	83	Female	Hypertrophic Arthritis	Chronic	Undetermined
15.	57	Female	Neuritis	Chronic	Undetermined
16.	26	Female	Muscular	Chronic	Undetermined
17.	71	Male	Hypertrophic Arthritis	Chronic	Traumatic
18.	54	Female	Neuritis	Acute	Undetermined
19.	35	Male	Sciatica	Acute	Traumatic
20.	47	Male	Neuritis	Chronic	Infected
21.	53	Male	Sciatica	Acute	Infected
22.	50	Male	Sciatica	Acute	Infected

In summarizing the results obtained in this series, those getting complete relief from pain will be classed as marked improvement, those showing considerable relief from pain but not completely relieved will be classed as improved.

The group showing improvement also included a few patients who obtained complete relief for a period of several weeks but had some recurrence of pain and returned for further treatment. There were two patients who did not show any improvement. One was the neuritis associated with pregnancy, mentioned previously, the other fell and injured her back and could not continue treatment.

SUMMARY

1. In this group of 22 patients treated with an injectable form of bee venom,* eleven showed marked improvement, nine were considerably improved, and two did not show any improvement.

2. No form of complimentary treatment was used except an attempt to improve the general health of the patient by proper diet and vitamin intake.

3. It would seem from the foregoing statements that bee venom is worthy of further consideration in the treatment of arthritis and neuritis.

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*Sharp and Dohme's "Lyovac."

SOME NEW INSTRUMENTS AND TECHNIQUE IN OPHTHALMIC SURGERY

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Puncture, counterpuncture, and section of about one-half of the corneal circumference in an upward direction is routine practice in extraction of cataracts.

One reason that is given for a primary incision upward is that the iridectomy will also be upward, and that less glare and color fringe disturbance will occur with iris defect partly covered by the upper lid. Secondly, it is said that the cosmetic deformity from an iridectomy upward is naturally less than from one placed laterally or downward. Thirdly, it is alleged that with the incision above, the upper lid affords the best kind of coverage and protection for the wound until healing becomes complete.

I enumerate the three points of the preceding paragraph as matters of general acceptance and belief. However, it is well known that upward iridectomy does not guarantee against glare or color fringe disturbance; and cosmetic deformity is of minor importance as compared to assuredness of a good functional result. Regarding the third point, if there is any value in coverage of the wound by the upper lid, it is strange that prolapses of iris and bulgings of the wound occur as often as they do, and that surgeons find a need to employ corneal sutures, conjunctival bridges and flap types of operations. And while it is true that the eyeball is rotated upward in sleep, at which time it is possible that the upper lid covers the entire cornea, in the waking hours of the patient it is probable that the eyeball beneath the bandaged lids frequently directs itself straight forward or slightly downward, in which event the line of the wound is approximately in line with the lid margin. The main part of upper lid pressure is then exerted upon the sclera above the wound, and little or none upon the loosened cornea below, on which account separation of wound margins, and serious complications result.

In additional disfavor to the upward incision are: First, the unhandy working position of the surgeon whether in back of the patient's head or on his right side in case of operating on the left eye. Second, the difficulty in deep set eyes of not stabbing or cutting the lids, and of obtaining access to sufficient area of conjunctiva to form an adequate flap or bridge. Third, the excessive drag upon and distortion of the eyeball in completing the corneal section, that predisposes to zonular rupture and vitreous loss. Fourth, the great danger of vitreous loss in presence of iridic adhesions.

These foregoing considerations prompted me to study the possibility of achieving a greater degree of safety in, and a more facile execution of, cataract and other ophthalmic operations.

Therefore during the past several years, I have devoted my spare energy to devise certain new instruments and with these instruments to test out various operative techniques on the eyes of live rabbits, and I am now in process of applying my observations to the human.

The management of rabbits for this type of study is of some incidental interest. I find them to handle best under drug anesthesia; that is, I inject dosages of 1½ grains of nembutal in 10% alcohol and water solution subcutaneously. In some rabbits one dose is sufficient; in others a second or a third at half-hour intervals are necessary. After induction of sleep, local injections of 2 per cent. procaine with 1-30,000 epinephrin are used in the lids, conjunctiva and retrobulbar tissues to render the eyeball and its adnexa insensitive. The so-called noval anestubes are employed with a dental type injecting syringe. For ease of access while operating, I dislocate the eyeballs from their sockets by means of a two-pronged fork fashioned out of a piece of drill wire.

Modern eye surgery involves with increasing frequency the loosening of the conjunctiva from the sclera with the creation of divers pockets, bridges and flaps. This loosening has usually been effected by spreading beneath the conjunctiva the blades of a small curved squint scissors after primary incision. The Woodruff elevator is another instrument that is used for stripping back the conjunctiva close to the cornea and also for splitting the cornea in the trephine operation for glaucoma.

The elevator of my own design is one that fulfills its purpose without danger of tears or perforation, and with admirable speed and efficiency. Observation of the cut will make the details of its construction clearer.

It will be noticed first, that the blade of the instrument is bent at about a right angle to the shank. Thus it is easily manipulated over any part of the bulbar surface, because motion of shank and handle of the instrument is out of the way of hindrance by either eyelids or bony orbital margins. Second, the blade is concaved on its under surface longitudinally and transversely for perfect adaptation to the scleral contour. Third, on this same blade the two sides and the broadened terminal edge, ground fairly sharp, are also concaved. These concavities favor easy separation of the conjunctiva around the bend of the corneal limbus.

The second instrument that I have devised is a single edged keratome. The standard two edged variety is poor in penetrating quality and has a tendency to bind or lock in the wound. Furthermore, if one is going to avoid injuring the lens, he must so direct the point of the instrument as to go through the cornea quite obliquely, leaving a shelf of tissue that overhangs the root of the iris; an obnoxious hindrance in peripheral iridectomy and iridotasis procedures.

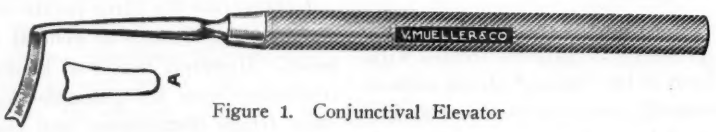

Figure 1. Conjunctival Elevator

Corneal Bistoury and Single Edged Keratome

As seen in the cut, the blade of my instrument joins the shank at an angle of about 30 degrees. It is concaved on its under surface for adaptation to the scleral contour. The noncutting back edge is broadly curved. The cutting edge forward and opposite is slightly concaved. After puncturing with this instrument the point is directed sideways into the irido-corneal angle. There is no risk of nicking the lens, the line of incision is kept truly marginal, and the iris angle is exposed in its depth. This blade is excellent in penetrating quality without tendency to bind or lock in the wound. Among other things it is perfectly adapted through its concaved under surface to shave from the cornea the apex of a pterygium.

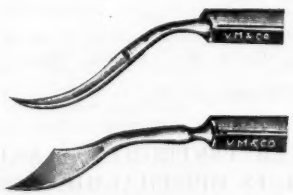

Figure 2. Corneal Bistoury and Single Edged Keratome

My third instrument which I call a corneal

bistoury has a thin sickle shaped blade, bent on the flat to an angle of 45 degrees with the shank. With it very delicate and skillful maneuvering is possible to effect a truly marginal opening into the irido-corneal angle. The small primary opening can be enlarged to any desired extent with fine curved scissors.

A case recently presented itself to me in the person of a woman eighty-three years of age, regardless of her years in good general physical condition. She had bilateral cataracts, semi-mature in the left eye with vision of 20/200; completely mature in the right eye with vision reduced to light perception and projection. There were firm appearing adhesions between iris and upper portion of the lens. Here was an ideal case to test out the new instruments, and my idea of a correct operative attack. I extracted the cataract cleanly, without trace of vitreous loss via a temporo-lateral opening, maintaining the conjunctivo-corneal attachments at all times intact. The steps of my procedure were as follows:

1. Usual block infiltration with procaine epinephrin solution to induce anesthesia and motor paralysis of the lids.

2. Instillation of 4% cocaine and 1-1000 adrenalin drops alternately a few times, and a sub-conjunctival injection temporalward of the procaine epinephrin solution.

3. Slitting of the conjunctiva perpendicular to and temporalward from the cornea on the point of a cataract knife.

4. Loosening the conjunctiva from the sclera on each side with the conjunctival elevator, working well up to and around the curve of the corneal limbus.

5. Puncturing the limbus with the bistoury knife (single edged keratome will do as well) in the line of the conjunctival slit.

6. Enlargement of the puncture to left and right in the line of the limbus with curved squint scissors, the one blade being within the anterior chamber in the irido-corneal angle, the other outside the sclera but beneath the conjunctiva. The cornea detached from sclera in about two-fifths of its circumference, nevertheless remains attached at all points to the conjunctiva.

7. Lateral small iridectomy, using U. S. Army model angular iris forceps and DeWecker scissors.

8. Transverse capsulotomy.

9. Expression. Gentle spoon pressure is exerted upon the corneal surface nasalward. At the same time the cut in the limbus is held open by traction with a delicate T shaped retractor on the bands of conjunctiva adjoining the conjunctival slit.

10. Replacement of any displaced iris after lens delivery with spatula. Sewing shut of the conjunctiva with a single running suture of fine black silk. Tying the two thread ends to one another over the course of the running suture.

11. Atropine instillation. Monocular dressing and bandage placed on the eye operated on only. Redressing and bandaging of the eye on the third day, at which time the patient was allowed home and to stay out of bed. Suture was removed on the sixth day.

12. End-result. There is a perfectly clear pupil, with lateral coloboma, completely free of opaque secondary membrane. Vision with correction is 20/40, which is sufficient to make the old lady comfortable and happy and able easily to get around. With reading correction she readily discerns fine print. There is some glare disturbance on account of the lateral coloboma which is not serious. I believe that a lateral iridotomy would be preferable to iridectomy for eliminating completely both coloboma and glare.

SUMMARY AND CONCLUSION

The upward incision is an important cause of failures in cataract surgery. A case is presented of extraction of the lens lateralward beneath conjunctival flaps. The pressure of both lid margins transversely across the center of the wound in this form of operation is a better safeguard against prolapse. Ease of execution, monocular occlusion and much shortened hospitalization are further advantages. New instruments are offered to facilitate this and other types of ophthalmic surgery.

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CONSTIPATION

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Constipation may be roughly defined as an impairment in the inherent capacity of the colon to produce formed stools and empty itself completely at regular and frequent intervals. We are still looking for a cure for constipation. Each announcement of a new drug, therapy or what not, is rapidly absorbed by both the profession and the layity, soon to fall by the wayside and be replaced by another innovation. The reason there are so many cures offered for constipation and so many medications on the market is because there is no one cure. The fact of the matter is there never will be a single cure, because there is no single cause for constipation.

ETIOLOGY

The etiology of constipation is often complex and may be due to one or more of the following causes:

Functional Causes

Disregard for habit time.

Sedentary habits and lack of exercise.

Insufficient water intake.
Dietetic deficiencies (bulk, vitamines and mineral salts).
Instability of the vegetative nervous system.
Endocrine dysfunctions.
Functional dyschesia (rectal constipation).
Coprostasophobia.
Psychic and nervous factors.

Organic Causes

Kinks.
Adhesions.
Anomalies of the colon.
Weakness of the abdominal muscles.
Diverticulitis.
Intrinsic and extrinsic tumors of the rectum and colon.
Hirschsprung's disease.
Gastroenteroptosis.
Rectal and sigmoidal prolapse.
Organic Dyschesia (rectal constipation).
Anal pathology (especially pectenosis, fissure, cryptitis and anal stenosis).

FUNCTIONAL CAUSES

Disregard for habit time

Perhaps the greatest perpetuating cause of constipation is the disregard for habit time. Much of the trouble is due to early neglect of the bowels. Getting up too late, rushing to school, shop, or office, and later neglecting the call of nature because of preoccupation, laziness, or embarrassment, have frequently been pointed out in this connection. To this should be added the uninviting condition of toilets in the poorer types of schools, shops, offices, farms and tenements.

Sedentary habits and lack of exercise

Although examples have been cited of riders who are frequently constipated and shut-ins who are not, it is generally conceded by physicians as well as laymen that exercise usually aids in regularity. Certainly building up the abdominal muscles aids materially in increasing intra-abdominal pressure which is an important factor in normal defecation.

Insufficient water intake

A common dietetic error is the consumption of a highly concentrated dry diet. With the exception of an occasional dish of soup or cup of coffee, many persons consume practically no fluids (specially during the winter) from one day's end to another. In such cases, the drinking of water on a fasting stomach in the morning as well as before meals, may be all that is necessary to cure the constipation.

Dietetic deficiencies

A lack of roughage, vitamines and mineral salts in the general American diet of meat, potatoes, coffee and pie is another common offender. Lack of sufficient roughage for those with atonic colons, and an inability to eat roughage in cases of spastic colon, gall-bladder disease and peptic ulcer contributes to constipation Spark and Collins; Morgan and Berry and others, have shown experimentally and clinically that vitamine B is necessary for the maintainance of normal muscular tone of the large intestine. Robertson and Doyle have shown that calcium and phosphorous deficiencies are capable of producing atonic colons. Also, obese women who are constantly dieting, eating one or two small feedings a day are not taking in sufficient bulk to produce normal daily evacuation. A good rule to keep in mind, therefore, is "no regular stool output without regular food intake."

Instability of the vegetative nervous system

The stress and strain associated with the demands of modern civilization plus the American habit of hustle and bustle has produced one of the diseases of modern civilization, namely, the unstable colon. Constipation manifests itself on the soil which has been prepared through the agency of an unstable nervous system. This may be congenital or acquired.

Endocrine Dysfunctions

Hypothyroidism—Mild Myxedema

In women with low metabolic rates, in the fourth and fifth decades, frequently of the obese type, there is a general lowering of endocrine glandular function, probably associated with ovarian dysfunction. Where intractable constipation exists with the above it may be suspected that hypothyroidism or mild myxedema exists (Brown).

Hypopituitarism

It has been demonstrated that pituitary preparations stimulate intestinal peristalsis (Ambros); therefore it is not illogical to assume that lack of pituitrin secretion will also contribute to some type of constipation.

Functional dyschesia (rectal constipation)

In some cases of constipation it is found that the rectum is practically always filled with feces. Study by means of the roentgen ray shows that there is no delay in the progress of the stool through the proximal colon. In other words, we are dealing with a condition in which that portion of the gut charged with the actual act of defecation fails to function. The pathogenesis of this disorder, known as dyschesia, has been described by Hurst as follows: "The habitual neglect of the call to defecation leads to the accumulation of the feces in the rectum and pelvic colon which gradually become more and more distended. The distention diminishes the tone and impairs the contractibility of the musculature; as the force required to empty it under normal conditions, the weakened muscular coat is incompetent to do its work, and even, if a greater effort be made, the evacuation remains incomplete. In time, the musculature of the pelvic colon and rectum may become so profoundly atonic and paretic that it can never be restored to its normal condition."

Coprostasophobia

There are individuals who constantly feel that there is something in the rectum which should come out. Some really suffer from rectal constipation; while others are just "hep" on the subject of rectal hygiene and are forever cleaning themselves out.

Psychic and nervous factors

The importance of psychic factors in constipation can hardly be exaggerated. Ever so many people know from experience that they cannot possibly have a bowel movement when nervous or worried, or while actively engaged in the ardent pursuit of their daily occupation. Even mild excitation, such as that associated with travel and change of environment, is a potent cause of constipation in susceptible individuals, as are, of course, the grosser emotions of fright and anxiety. Fatigue may also be considered under the head of nervous factors. A sufficient degree of general bodily rest and relaxation is essential to the proper functioning of all the organs and the bowels form no exception to the rule. Whether or not the fatigue products of metabolism are actually constipating, it is nevertheless certain that many cases of costiveness are associated with insufficient rest, and particularly with insufficient sleep. This is very often the case with those who work at night.

ORGANIC CAUSES

Kinks

At certain points, kinks are apt to occur when there is a dropping of the stomach and intestine. From above downward, kinking may occur at the junction of the duodenum and jejunum, at the distal end of the ileum, at the hepatic flexure, at the splenic flexure and at the sigmoid flexure. Should pericolic membranes form around these kinks an actual complete obstruction may occur.

Adhesions

Intestinal interlining adhesions produced by an exudate upon the intestinal wall as a result of the activities of putrefactive microorganisms have been described. These adhesions aided by angulation or colonic spasm can distort the colon into all sorts of shapes, producing pockets of various dimensions, as well as constrictions able to cause serious strangulation. Case quotes Eastman, Hartzler and Jackson who have shown that it is possible for extensive colonic adhesions to exist as the result of chronic intestinal stasis, when we are able to elicit no history pointing to the existence of any previous intestinal inflammation. It must be kept in mind however, that functional atonicity, spasticity, dyschesia, etc., may be coincidentally present and responsible for the constipation instead of the also existing kinks, membranes, adhesions, etc.

Anomalies of the colon

The most important congenital anomaly contributing to constipation is redundancy of the colon. Over three-quarters of the patients with this malformation suffer from intestinal stasis. One should not imagine that the redundant loops cause actual obstruction, but rather that they furnish the site for the development of localized spasms, and pockets, retaining old fecal debris and voiding only the overflow.

Redundant colon was encountered in almost 25 per cent. of the writer's series of over 612 patients complaining of constipation. The redundant colon occurs more often in men and in the sthenic (stout) habitus. It is, as the name implies, a condition of increased colonic length. The redundancy may take the form of simple

pleats or reduplications or of extensive loops and twists. The most frequent site is the pelvic colon. The condition is believed to become exaggerated following chronic rectal constipation and straining at stool.

The constipation of colonic redundancy often dates back to childhood. The interval between spontaneous bowel movements may extend from three days to a week or more. There is often discomfort in the lower right quandrant, characterized as dull, dragging, or sticking in nature, and increased by bodily exertion.

Not infrequently there is gas pressure, fullness or pain referred to the exact location of the redundant loop in the left colon. Sometimes precordial distress or increased belching is provoked by gas accumulation under the left diaphragm. The passage of flatus is at times difficult, but when it does take place, is often followed by complete relief of symptoms.

Normally the cecum is unattached, whereas the ascending colon is fixed to the posterior abdominal wall. If the normal fixation of the ascending colon is continued downward, the cecum becomes fixed in its turn to the posterior abdominal wall. With this anomaly we frequently have a very low cecum which is fixed producing the condition known as "low fixed cecum." Low fixed cecum is most commonly present in women of the asthenic (tall) type. This is the condition that frequently receives the diagnosis of "chronic appendicitis." Low fixed cecum is one of the anomalies responsible for constipation.

Weakness of the abdominal muscles

Weakness of the abdominal muscles following childbirth, obesity, abdominal operations, advancing age, diastasis recti, and lack of abdominal exercise diminish intra-abdominal pressure, an important factor in producing normal bowel evacuation.

Diverticulitis (especially the hyperplastic variety)

Diverticulitis producing a hyperplastic tumefaction will progressively narrow the lumen of the affected bowel and cause a progressive constipation. Simple diverticulitis is usually associated with spastic constipation.

Intrinsic and Extrinsic tumors of the rectum and colon

Carcinoma of the rectum and colon may produce progressive obliteration of the bowel lumen with progressive constipation. Also, pressure on portions of the sigmoid and colon from tumors in adjoining organs such as fibroids, ovarian cysts, carcinoma of the uterus, Krukenburg tumors and endometriosis. Strictures of the rectum due to lymphopathia venerea, syphilis and ulcerative colitis are also causes of constipation.

Hirschsprung's disease—(Megacolon)

Megacolon is a congenital or acquired dilation and hypertrophy of the colon, characterized by bowel movements once in one to three weeks, abdominal distension, intestinal toxemia, etc. Buie suggested the following causes: 1. Congenital dilatation and hypertrophy of the colon. 2. Abnormal length and loop formation of the sigmoid flexure. 3. Idiopathic dilatation of the colon. 4. Spasm of the sphincter ani. In the diagnosis and treatment of constipation one must be on the lookout for this condition especially in children with distended abdomen.

Gastroenteroptosis

There is no longer any question that body habitus exerts a definite influence on gastrointestinal motility. For example, in sthenic (stocky) individuals, the bowels normally tend to be free, moving spontaneously as often as two or three times daily. On the other hand, in asthenic (slim) persons, the bowels normally move but once a day, or perhaps less often. This is not to say that visceroptosis is invariably associated with constipation, or that constipation in asthenics can only be relieved by correcting the ptosis, or even that correction of the ptosis alone is invariably followed by relief of the constipation. The fact remains, however, that constipation is more common in the asthenic (tall) type than in the sthenic (stocky) and that abdominal support is usually beneficial in ptotic constipated patients.

Rectal and sigmoidal prolapse

A condition very seldom mentioned by writers as a cause of constipation is rectal and sigmoidal redundancy. Here we have an excessive amount of rectal and sigmoidal tissue which causes an invagination or partial prolapse on bearing down at stool.

Instead of pushing stool down by the mechanism of defecation, the bowel wall is forced down instead, obstructing the passage of the stool. This condition is diagnosed during digital palpation by asking the patient to bear down, when

the bowel will be felt to come down and meet the examining finger.

Organic dyschesia

Organic dyschesia frequently develops secondarily to any condition which causes pain during defecation, such as inflamed or thrombosed hemorrhoids, anal ulcers or fissures, pectenosis and pelvic peritonitis. In women, the condition is frequently caused or aggravated by childbirth. Special cord lesions may cause a stubborn dyschesia.

Anal pathology

Such as fissures, cryptitis, papillitis, pectenosis and anal stenosis are common causes of constipation, frequently overlooked.

COMMON TYPES OF CONSTIPATION

In 1912 Gustav Schwartz divided functional constipation into four types:

1. Ascending type.
2. Hypokinetic type (atonic colon).
3. Dyskinetic type (spastic colon).
4. Dyschesia.

Ascending type

In this type a constriction forms at the neuromuscular ring of Keith in the transverse colon and interferes with the forward propulsion of the intestinal contents. This is not a very common type of constipation.

Hypokinetic type (atonic colon)

This type involves the entire colon. It may be due to hypoactivity of the vagus or hyperactivity of the sympathetics, and is usually associated with hypoendocrine activity as in the hypothyroidism and hypopituitarism types. This type usually occurs past menopause and is less frequent than the spastic variety.

Dyskinetic type (spastic colon)

This is characterized by spasm in the descending colon and sigmoid usually due to parasympathetic hyperactivity. This, perhaps is the most common variety of the disease and is present in about 90 per cent. of the cases of constipation. When spastic constipation is associated with atony of the proximal large intestine, the cecum, ascending colon and transverse colon act as reservoirs for large accumulations of feces which are passed, small portions at a time, beyond the contracted areas. Clinically, this condition may be associated with daily, but quite insufficient evacuations. The common symptoms of spastic constipation are abdominal pain, generally localized in the left lower quadrant, often increased by defecation, and still more by catharsis; peristaltic unrest, and the passage of small stools, hard and lumpy in character (marble or sheep-dung stools). Examination of the left lower quadrant often reveals a tight cord corresponding to the iliac colon. The ceco-colon is often distended, gurgling and tender. The anus may feel tight to the examining finger while inspection of the pelvic colon with the sigmoidoscope often reveals the presence of an irritable condition which may prevent the introduction of the whole length of the instrument.

Functional Dyschesia (rectal constipation)

This type of constipation is due to a loss or diminution of the defecation reflex in the rectum and is usually diagnosed by the examining finger which locates the presence of stool in the rectum in a patient who has no desire to defecate. Or it may be due to some painful lesion in the anal canal such as a fissure, cryptitis, papillitis, pectenosis, stricture and anal stenosis.

DIAGNOSIS OF CONSTIPATION

If the patient complains only of constipation without any abdominal pain or belly-consciousness the findings point to a hypokinetic (atonic) type of constipation.

If on the other hand the constipation is accompanied by abdominal pain, excessive mucus, and increased discomfort when taking laxatives, the condition is usually dyskinetic or spastic constipation.

Progressive constipation with abdominal discomfort may be due to hyperplastic diverticulitis, tumor in the left colon, stricture, carcinoma of the rectum, as well as to extrinsic pathology such as Krukenburg tumors, fibroids of the uterus, ovarian cysts, Hodgkin's disease, presacral dermoids, etc. Proctoscopic and sigmoidoscopic examinations are very important in every case of constipation. Any recent change in bowel habit or abdominal cramps in a person past forty should be suspected for carcinoma until proved otherwise. Occult blood, bloody mucus or black stools suggest malignancy as a cause of constipation.

If the patient is obese and the metabolism is low hypothyroidism should be suspected.

The carmine test or the barium meal roentgen

examination are very excellent aids in the determination of an existing constipation. Barium retention in the colon after 36 hours suggests constipation. We have seen barium retention in a patient as late as 144 hours (six days). The longer the retention the greater the degree of constipation. If the barium meal is arrested at about the mid-transverse colon it suggests the ascending type of constipation. If the left side of the colon appears narrow and stringy, spastic colon exists and if the entire colon seems dilated, atonic colon can be diagnosed. By administering two 5-grain capsules of carmine with breakfast the red color in the stool should appear normally in 12 hours and disappear in twenty-four hours. This is an excellent aid when x-ray is not available or as an inexpensive check-up from time to time to watch the progress of treatment. Physical examination is helpful and is performed by abdominal palpation and by rectal touch. By the former method, a contracted or knotted and usually tender iliac colon may be rolled over the left iliac fossa in cases of spastic constipation. In atonic constipation the entire colon is dilated and is recognized by its lack of tone. The metabolism is frequently low. In dyschesia a rectum filled with hard scyballa may be discovered by the gloved finger even though the patient has no desire to go to stool. If the anal canal is very painful it suggests anal pathology which is contributing to the constipation.

TREATMENT OF CONSTIPATION

Prophylactic treatment

Prophylaxis should provide for regular food intake, for routine visits to the toilet, sufficient water drinking especially on arising, a balanced dietary containing sufficient roughage, abdominal exercises and correct posture.

General treatment

Habit time

Patients should be instructed to go to stool at the same hour each day, either before or after breakfast; to try for ten minutes by straining at regular intervals; to omit this if hypertension, hernia or anorectal disease exist. If no result is obtained to insert a glycerine suppository.

Enemata

If no result with the above, patient is to take a glycerine enema consisting of one to three ounces of glycerine to a pint of water. In rectal (dyschesia) and sigmoidal constipation it is betterto give an enema than to disturb the entire intestinal tract by a laxative. If anal pathology exists omit the glycerine.

Toilet facilities

Boles has pointed out that the children of China, India and Japan rarely have constipation. This is because these children from infancy on are taught to defecate in the squatting position with the thighs flexed on the abdomen. The adults continue this habit by squatting over a hole during defecation. To duplicate the squatting position, a foot-stool can be placed in front of the toilet (in any home) to raise the knees and flex the thighs on the abdomen. There is evidence that plumbing manufacturers are already building low toilet bowls which tend to simulate the oriental hole technic.

Water intake

About eight glasses of water should be consumed in a day. Drinking normal saline water or lemon juice and water on arising will frequently in itself produce the act of defecation.

Diet

Irregular eating habits are a common cause of constipation. "No regular stool output without regular food intake," should be preached to patients.

Simple starvation or inordinate food restriction is also a common cause of constipation. In some cases, there is an actual physical cause preventing the consumption of normal amounts of food, such as an obstruction in the digestive pathway, or a painful lesion which causes fear of eating. In many individuals, however, various articles of diet are progressively eliminated in an awkward attempt to cure the very constipation that is aggravated by the process. Obviously, the remedy for this state of affairs is the early restoration of an enlarged and balanced dietary.

It is a matter of common knowledge that certain individuals habitually omit from their diet those foodstuffs possessing inherent qualities of increasing intestinal peristalsis. Such foods include the vegetables and cereals with cellulose residues which stimulates peristalsis chiefly by mechanical means; the fruits which also act mechanically, and the fats which act both chemically and mechanically.

Patients on ulcer diet, spastic colon diet, gall-

bladder diet and obesity diets become constipated and should have some form of smooth bulk added to their food lists such as karaya, ground psyllium seeds, bassorin, agar agar, sterculia gum, etc. (see Gums).

Coprostasophobia and Psychotherapy

In cases of coprostasophobia where all investigations prove negative, patients should be shown the x-ray films and results of other tests to convince them of their normalcy. The psychic and nervous causes of constipation should be controlled as much as possible by appropriate mental and emotional hygiene, by adequate vacations, rest, and sufficient sleep, and by sedative medication.

Visceroptosis and Malnutrition

Should receive appropriate attention. Abdominal support and a fattening diet should be prescribed when indicated. For the latter, butter, cream and olive oil are particularly to be recommended.

Abdominal exercise

Is helpful because it tightens the abdominal wall and increases intra-abdominal pressure.

Gums (Smooth bulk producers)

Mechanical laxatives are definitely replacing chemical laxatives chiefly becaues they produce smooth bulk and lubrication without chemical stimulation, irritation or absorption. The more commonly used gums are agar agar, bassorin, karaya, sterculia gum and psyllium seed.

Whole psyllium seed is diminishing in popularity because of its tendency to cause impactions, but the granulated and purified forms are quite satisfactory. Ivy has found that karaya is not digested in the intestinal tract, is not affected by B. welchii, does not produce gas or fermentation, does not effect nitrogen utilization in therapeutic doses, does not absorb vitamin A, is not irritating and is not habit forming. Gum laxatives are taken in one or two dram doses once or twice a day followed by two glasses of water. Patients who are reducing and wish to curb their food intake should take the gums before meals. This helps to fill the stomach and curb hunger. Otherwise it can be taken after meals.

Medicinals

In atonic constipation strychnine 1/60 gr. to 1/30 gr. given about one hour before breakfast will increase the reflex irritability of the bowel by the end of the meal (Mulinos). 1/150 gr. to 1/200 gr. atropine or 1/6 gr. to 1/4 gr. extract belladonna or 1/6 to 1/2 gr. extract hyoscyamus, paralyze the myoneural junctions of the parasympathetics and are beneficial in spastic constipation. Cascara in some form and in varying doses is good therapy to add to gum laxatives when necessary. Bassorin and mucara are both gum laxative compounds with cascara. Siblin is an excellent smooth bulk laxative containing vitamine B.

Phenolpthalein

Is contained in more than 125 proprietary preparations in the form of laxative drugs, chewing gums, confections, fruits and biscuits. It is believed to produce intestinal catarrh, skin eruptions and visceral disturbances in sensitive patients and is best omitted in the treatment of constipation (Newman).

Mineral oil

Is diminishing in popularity because it mixes with food and interferes with digestion, absorbs some of the vitamins, tends to leak and increases intestinal flatus.

Lacto-dextrin, acidophilus and lactic acid preparations

Of any kind frequently increase spasticity by increasing the acid content of the bowel; do not correct constipation in the majority of cases and lose their flora-changing effect soon after the preparation is discontinued.

Colloidal-Kaolin

A colloidal form of refined Fuller's earth has the property to absorb and adsorb bacteria and toxins in the intestinal tract and render them innocuous. It is inert and is not absorbed in the intestinal tract. It soothes the mucous membrane and aids in changing the intestinal flora. It does not produce reactions and aggravations of symptoms as is commonly noted with the lactic acid preparations. It has no contraindications. In the writer's hands it has been a most satisfactory remedy in the treatment of many gastro-intestinal disturbances. Two drams to two ounces a day dissolved in water can be given without reactions. For changing the intestinal flora two ounces in divided doses at first, cutting down to one dram twice a day is excellent treatment.

Colloidal kaolin in combination with phenobarbital gr. ¼; extract of hyoscyamus gr. ½ and magnesium oxide in varying doses(Maolin formula No. 3) has been the writer's most satisfactory compound i nthe treatment of spastic colon.

Bran

Is rough, tends to pack, and is contraindicated in spastic colon, gall-bladder disease, ulcerative lesions of the intestinal tract and anorectal disease.

Vitamines B and G

Is believed to increase or normalize colon tone by its effect on the vegetative nervous system; or by the favorable effect of increased food intake following increased appetitie, produced by these vitamines.

Mineral Salts

Mineral salts, especially calcium and phosphorus, should be added to the dietary in every case of constipation, especially the atonic variety (Robertson).

Endocrine therapy

According to Lahey only those of the hypothyroidism group (mild myxedema) with a blood cholesterosis of 200 or more will respond to 2 to 6 grains of thyroid daily. Weiss has suggested multiple therapy which includes vitamines B and G, lacto-banana concentrate, trilactic, bile extract, hormone cholezysmon, duodenal extract and agar gum karaya. Ambrose reported several cases of chronic constiption which were successfully treated with pituitrin after other measures had failed.

Surgery.

The most indicated surgery in the treatment of constipation is that of the anal canal. Conditions such as cryptitis, fissure, pectenosis and anal stenosis should be corrected. The repair of ventral hernia and diastasis recti are also aids in increasing intra-abdominal pressure and correcting constipation. Removal of a chronic appendix will frequently aid in correcting a spastic colon. Operations for the relief of visceroptosis, redundant colon, adhesions, bands, kinks, etc., in the treatment of constipation should only be done as a last resort and then only after due consideration and deliberation. Resections of the colon for infectious granulomata and carcinomata are indicated and necessary. Surgical removal of a hyperplastic diverticulous tumefaction is rarely necessary. Removal of tumors pressing on the bowel such as fibroids, ovarian tumors, endometriosis, presacral tumors, etc., are indicated and necessary to relieve extrinsic causes of constipation. Sympathetic ramisection to relieve hyperactivity of the sympathetic innervation of the rectum or rectosigmoid sphincter has proven beneficial in selected cases of Hirschsprung's constipation (Buig. Royle).

TREATMENT OF THE MOST COMMON FORMS OF CONSTIPATION

Spastic constipation

Is successfully treated by rest, sedatives, intestinal antiseptics, antispasmodics and non-irritating mild laxatives. Such a combination is Maolin made up of colloidal kaolin, magnesium oxide, phenobarbital and extract of hyoscyamus previously mentioned. The amounts can be varied according to the needs of the patient. Where smooth bulk is desirable any of the gums previously mentioned plus the following combination works well—

℞

Powd. extra. of hyoscyamus . gr. 1/4
Powd. extra. of belladonna . gr. 1/6
Phenobarbital ..... gr. 1/4 to gr. 1/2
Powd. extra. of cascara .... gr. 1/6 up (can be added if necessary)
Misce, fiat. caps. No. 1.
Sig. 1 caps. 2 or 3 times a day.

The diet should be bland to help put the colon at rest. Cleansing enemas of normal saline and sodium bicarbonate solution are helpful to start off the treatment and then taken only occasionally as necessary. Oil retention enemas can be used if the stools are very dry, but is a nuisance and seldom if ever adhered to by patients for any lengthy period. Estrogenic substances are helpful in spastic colon cases going through the menopause. Psychotherapy is frequently indicated. Appendectomy sometimes helps when other things fail. Removal of anorectal pathological changes is most important in many cases.

Atonic constipation

Is best treated by stimulating peristalsis in several ways. First a liberal mixed diet including raw vegetables and raw fruits unless contraindicated by the coexistence of gall-bladder dis-

ease. In addition the smooth bulky gums with an abundance of water twice a day. Abdominal exercises to increase intra-abdominal pressure, strychnine 1/60 gr. to 1/30 gr. and thyroid extract gr. ½ and up three times a day are all helpful aids to stimulate peristalsis in atonic constipation. Glycerine enemata as suggested previously to start the treatment off and then as necessary is helpful.

Dyschesia

In the treatment of dyschesia all anal pathology such as fissures, cryptitis, papillitis, pectenosis, etc., should first be alleviated. Glycerine enemata or glycerine suppositories are more satisfactory than oral laxatives. A suppository containing 3 to 5 grains of quinine dihydrochloride in oil of theobroma (low melting point) helps to renew or restore the desensitized defecation reflex (Graham); 25 cc. of this same drug in a 2 per cent. solution, absorbed in cotton balls and placed into the rectum through a proctoscope three times a week is helpful. Patients usually discharge them by the following morning. It is important to clear up all anal pathology before using this drug.

In rectal and sigmoidal mucosal redundancy one or two things are indicated. Either a sigmoidopexy, pulling the loose bowel up from above and fixing it to the abdominal wall or any other of the recognized procedures; or to inject 4 percent. solution of urea-quinine hydrochloride in the redundant bowel wall through a sigmoidoscope.

CONCLUSIONS

The causes of constipation are many and varied. An outline of functional and organic causes are discussed.

A complete history, physical examination, metabolism determination, x-ray, stool analysis, and proctologic examination are necessary to establish the cause of constipation.

The general treatment and specific treatment of constipation is presented.

30 N. Michigan Blvd.

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THE DELETERIOUS EFFECTS OF SULFANILAMIDE AND SULFAPYRIDINE

With a Case Report of Agranulocytosis

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Since the disagreeable and harmful effects of sulfanilamide and sulfapyridine are very similar they will be discussed under the same general outline, and because very little can be found in the literature regarding any harmful effects of the third member of the sulffanilamide group i.e. neoprontosil, only occasional mention will be made of this drug.

These disagreeable and sometimes dangerous reactions may be divided into the following groups:

I. *Cyanosis*:

This condition is likely to begin very soon after the administration of either sulfanilamide or sulfapyridine and is of two types:

A. Methemoglobinemia. A condition arising from a chemical combination of the sulfone or SO_2 radical of these drugs with hemoglobin and preventing it from uniting with oxygen.

This condition was once considered to be a precursor of the anemias, but is not considered so at the preesnt time, and is said not to be particularly dangerous. It is usually well controlled by methelene blue, Gr. τ or π by mouth every 4 hrs. or more promptly by one milligram per kilogram weight given intravenously.

B. Sulfhemoglobinemia which is a cyanosis brought about by uniting of hydrogen sulfide

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with hemoglobin. This hydrogen sulfide may come from:

1. Breaking down of the sulfur radical from the sulfanilamide in the blood plasma.
2. Absorption of hydrogen sulfide from the bowel. Saline cathartics are said to increase this absorption as are the high sulfur foods such as beans, cheese, clams, cocoa and bran.

Methelene blue is used as a treatment and oxygen is valuable in more severe cases.

II. *Gastric Intolerance:*

The symptom of nausea or vomiting may be very pronounced and is caused by:

A. The local irritation of sulfanilamide and sulfapyridine on the gastric mucosa.

B. Acidosis produced by these drugs or the combination of infection and drugs which has not been properly combated by alkalis. Where the continuation of the drug is imperative, either sulfanilamide or sulfapyridine may be given rectally, sodium sulfapyridine given intravenously or sulfanilamide given subcutaneously, one or two per cent. in Ringer's Solution. Nicotinic acid given in doses of three to four hundred milligrams a day is said to be valuable in preventing this condition, as is also intravenous dextrose, alkalization, and barbiturates per rectum.

C. Diarrhea: This symptom is uncommon with sulfapyridine, occasional with sulfanilamide and frequent with neoprontosil, and if treatment is continued may produce marked inflammatory reaction with bloody stools. If this occurs sulfanilamide can often be substituted for neoprontosil or vice versa without difficulty. If the indication is urgent, bismuth subnitrate or some mild form of opium often permits continuance.

III. *Central Nervous System Reactions:*

These reactions may evidence themselves as:

A. Cerebral excitability with insomnia restlessness and even delirium.

B. Drowsiness or stupor.

C. Dizziness.

These symptoms if marked, indicate discontinuance or reducing the dosage of these drugs unless the indications are imperative.

IV. *Skin Reactions:*

These may assume any of the following forms:

A. Purpuric.

B. Urticarial.

C. Scarlitinal.

D. Erythematous with or without pustules.

E. Rubellaform.

F. Simple dermatitic with or without photosensitization.

G. Exfoliative dermatitic reaction.

Any of these reactions if severe may force the discontinuance of the drug. Especially is this true of the dermatitic group since these are often precursors of serious renal complications.

V. *Febrile Reactions:*

These may be immediate or delayed and are more likely to occur when these drugs are resumed after a short interval of discontinuance.

Unless the need is urgent febrile reactions indicate that either sulfanilamide or sulfapyridine should be immediately discontinued because this reaction is our best warning that hepatic, renal, or cellular blood reactions may likely occur shortly, if continued.

VI. *Peripheral Neuritis:*

This reaction may follow the use of either drug and may involve any nerve including the optic. It is believed that they are more likely to occur when sulfates are ingested with the drugs. Thiamin Chloride (Vit B_1) 10,000 Int. Units, subcutaneously every other day has been of some value in treating these cases.

VII. *Hepatic Reactions:*

A. Hemolytic Jaundice.

This condition occurs as a result of the overloading of the liver with hemoglobin pigments from the rapid disintegration of red blood cells and is characterized by nausea or vomiting, jaundice and bile in the urine. It is naturally a precursor of anemia and a valuable warning signal.

B. Toxic Hepatitis.

This is a reaction brought about by the toxic effect of sulfanilamide and sulfapyridine on the liver cells and is characterized by:

1. Nausea and Vomiting.
2. Jaundice.
3. Excessive bile in the urine.
4. Enlargement of the liver.
5. High bilirubin content of blood plasma.

This syndrome is more likely to occur with sulfanilamide than with sulfapyridine. It is frequently associated with dermatitis and is rapidly followed by renal complications. It is a definite contra indication to further use of these drugs.

VIII. *Anemias:*

These conditions are a result of the destructive effects of either drug on red blood corpuscles and may be divided into two types:

A. Mild simple anemia. This complication is characterized by a drop in both red cells and hemoglobin without red blood cell abnormality, is gradual in onset, usually occuring after several days of administration and more usually with sulfanilamide. As mentioned before it is associated with hemolytic jaundice, if marked. Large doses of iron (not the sulfate form) with Vit. B complex are often sufficient to counteract this form of anemia and permit the continued use of the drug.

B. Acute Hemolytic Anemia: This type of anemia often occurs early in the administration of sulfanilamide and rarely with sulfapyridine, being characterized by:

1. Rapid fall in red blood count and hemoglobin.
2. Macrocytosis and reticulocytosis.
3. Leukocytosis.
4. Jaundice and increased icterus index.
5. Blood in the urine and albuminuria.

Discontinuing the drug, transfusion, liver, iron and Vitamin B therapy are indicated.

IX. *Renal Complications:*

These conditions may be divided into the following groups:

A. Simple albuminuria without casts or blood cells and without nitrogen retention.

This condition clears up promptly with discontinuance or decreased dosage and forced fluids.

B. Nephritis: This is characterized by:

1. Albumin, blood and casts in the urine.
2. Anuria.
3. Increased N. P. N. of blood.
4. Edema.

This syndrome is much more likely to occur with sulfapyridine than with sulfanilamide administration, and especially when the fluid intake is too low and resultant blood stream concentration of the drug is above twelve milligrams per 100cc of blood. The fluid intake when giving sulfapyridine should not be below 3,000cc. Nephritis is a contra-indication to continued medication, unless the indication is very imperative.

C. Urinary Calculi: This complication occurs more often with sulfapyridine, but occasionally also in sulfanilamide therapy. It is characterized by the formation of crystals of either acteysulfapyridine or acetylsufonilamide in the ureters, renal pelvis and tubules. These concretions cause a dilatation of the ureters and renal pelvis, visible by x-ray examination, but with the concretions, which cause this dilatation, very difficult to demonstrate by x-ray.

They frequently, because of urinary obstruction cause, or are associated with hematuria, pyelitis and pyelonephritis.

These calculi occur when there is a decreased urinary output, and a consequent supersaturation of the urine with these drugs. A highly alkaline urine also favors their precipitation and forces us to question the routine administration of large doses of alkali without observation of the alkalinity of the urine, which should not be maintained above p.h.7.

X. *Agranulocytosis:*

This condition is one of the most formidable and dangerous of all these deleterious effects of both sulfanilamide and sulfapyridine, although perhaps more frequent with sulfapyridine. It occurs late and usually after two weeks of drug administration, the maximum amount so far reported previous to onset being about twenty-five grams of sulfanilamide and twenty-two grams of sulfapyridine.

It is caused by the depressing effect of the benzine ring contained in the drug on the blood forming tissues. It is characterized by:

A. Marked lowering of the white blood count with a relative decrease in polymorphonuclears cells.

B. Temperature, weakness and gastric distress.

C. Generalized gland enlargement.

D. Enlargement of liver and spleen.

E. Marked inflammation and edema of throat and mouth with white patches resembling the membrane of Vincent's angina and sometimes necrosis of the mucous membrane of mouth, throat or nose.

F. Blood, casts and albumin in the urine of the more severe cases.

This condition is treacherous because it may occur several days after the drug is discontinued with a white count above normal at the time of its discontinuance.

The treatment consists of immediate discontinuance of these drugs, pentnucleotide in maxi-

mum dosage, repeated transfusions, liver extract, vitamin B complex, forced fluids and symptomatic treatment.

The following case history is an example of one of these reactions.

REPORT OF CASE

A married male, age 24 with previous negative medical history was seen by me March 30, 1939, having become ill, with temperature and sore throat twenty-four hours previous to my visit.

The throat was very red with multiple white pearly patches on both tonsils, the tonsilar glands were markedly enlarged, the breath odor foul and temperature 101.5°. Sulfanilamide 90 grs. per day and sodium bicarbonate 60 grs. per day were prescribed.

March 31, 1939: Culture report negative for diptheria, but contained many long chain streptococci and fusiform bacilli. The maximum temperature was 100°, the white blood count 21,000, hemoglobin 85% (Talquist) and patient's general condition was improved.

April 2, 1939: The patient's general condition was improved, glands smaller, exudate in tonsils diminished and maximum temperature 99.5°. The white blood count was 11,000, hemoglobin 80%, but urinalysis showed quantities of bile, a few pus cells, numerous red blood cells and plus two albumin. Sulfanilamide reduced to 45 grs. daily.

April 4, 1939: The patient's general condition was worse with maximum temperature of 102° F, white blood count 15,000, hemoglobin 70%, but urine somewhat improved. Sulfanilamide was increased again to 90 grs. daily.

April 6, 1939: The patient was again improved with maximum temperature of 100° F, white blood count 11,400, hemoglobin 70%. The spots were gone from the throat, glands smaller and only a trace of bile and albumin in the urine. However, a rough systolic blowing sound was noticed in the heart, most evident at the apex. Sulfanilamide was reduced to 60 grs. daily.

April 8, 1939: The general condition was worse, maximum temperature 103.5°, white blood count 22,000, hemoglobin 65%, the urine still containing a trace of albumin and bile with a few red cells present. Sulfanilamide 90 grs. daily was resumed with the addition of Iron Ammonium Citrates Gr. 40, Vitamin B, 40 Int. Units and Ribrofavin Units 30, which were continued during the entire course of treatment.

April 10 to April 19, 1939: The patient improved gradually and on April 14, was temperature free, white blood count was 11,000, hemoglobin 80%, urine negative, glands almost normal and heart sound improved. The sulfanilamide was reduced to 40 grs. and discontinued April 15, having had a total of 1170 grs. or 78.4 grams. The patient remained temperature free until April 19, when he took a short automobile ride, but in the evening had a temperature of 100° F, with a recurrence of sore throat. By telephone he was instructed to take sulfanilamide Gr. X, every 4 hrs. until I could see him the next day.

April 20, 1939: The patient had a maximum temperature of 103° F, the glands were markedly enlarged, white blood count 3200, hemoglobin 75%. The throat was markedly injected with right retro tonsilar edema, but no exudate. The sulfanilamide was discontinued with the patient having had 42.5 grains since the previous evening.

April 21, 1939: The patient chilled after which the temperature arose to 105° F. He complained of pain in the precordial area and shortness of breath. The white blood count was 1600, hemoglobin 70%, and the red blood cells 3,900,000. A yellowish white exudate had appeared on both tonsils with foul breath odor.

April 22, 1939: The morning temperature was 103.5° F with the white blood cound 1400. The afternoon temperature was 104.5 F, with white blood count of 1200. Reticulogen 2cc and Vit B, 22,500 were given intramuscularly. The patient was admitted to the hospital that evening and at 8 P. M. had temperature of 106.4° F, red blood count 3,200,000, hemoglobin 60%, color index .9, white blood count of 1000 with differential; polymorphonuclears 1%, small lymphocytes 95% and large lymphocytes 4%. The urine contained an occasional pus cell and hyaline cast with one plus albumen.

April 23, 1939: The patient was transfused with 450 cc, by direct method at 4 A. M. The temperature was 105° F, with general gland adeno pathy, retrotonsilar edema of both tonsils, distant heart sounds with soft blowing systolic murmur and a choking sensation in the prone position. The red blood count was 2,800,000, hemoglobin 68%, color index .87 with white blood count 1000, all lymphocytes.

During the day 40 cc of pentnucleotide, 2cc of Reticulogen and 15000 Int. Units Vit B, were given and that evening 450 cc blood were given by indirect method. Throat culture showed long chain streptococci.

April 24, 1939: The maximum temperature was 104° F, hemoglobin 71%, color index .86, white blood count 1200, diffential; polymorphonuclears 2%, small lymphocytes 93%, large lymphocytes 4%, transtional cell 1%. Reticulogen 2 cc, Vit. B, 15000 and 40 cc pentnucleotide was given with transfusion of 550 cc (indirect).

April 25, 1939: Maximum temperature was 103.3° F, red blood cells 4,380,000, hemoglobin 77%, white blood count 1600, with differential; polymorphonuclear 8%, small lymphocytes 84%, large lymphocytes 6%, transitional cells 2%. Transfusion of 400 cc (indirect) was given and other medication continued.

April 26, 1939: The patient was much improved, the glands subsiding, breathing and heart sounds improved, maximum temperature was 101.4° F, red blood cells 5,050,000 hemoglobin 90%, color index .9, white blood count 4800 with differential; polymorphonuclear 45%, small lymphocytes 47%, large lymphocytes 6%, transitional cells 2%. The urine contained many red blood cells, a few pus cells, and one plus albumen. Transfusion of 320 cc (indirect) was given. The pentnucleotide was reduced to 30 cc, reticulogen to 1 cc and Vit. B intramuscularily discontinued. The mouth administration of Iron Ammonium Citrate and Vit. B were continued.

April 27, 1939: The maximum temperature was 98° F. The red blood count was 5,350,000, hemoglobin 100%, color index. .9%, white blood count 10,800 with polymorphonuclear cells 68%, small lymphocytes 26%, large lymphocytes 4%, transitional cells 2%. Treatment continued the same except for transfusion which was discontinued.

April 28, 1939: The maximum temperature was 99° F, red blood count 5,300,000, Hemoglobin 100%, white blood count 12,300 with polymorphonuclears 72%, small lymphocytes 21%, large lymphocytes 4%, transitional cells 3%.

April 30, 1939: The patient was discharged from the hospital with red blood count of 4,445,000, white count 18,300, polymorphonuclears 80%, small lmphocytes 18%, large lymphocytes 2%, and transitional 1%.

This patient has been observed many times since discharged and has a white count varying between nine and ten thousand with a normal differential. He has had his tonsils removed and suffered one mild attack of appendicitis (not operated on) at which time his white count did not exceed 12,000. He is clinically well and performing hard manual labor.

SUMMARY

Rules and precautions for the administration of sulfanilamide and sulfapyridine.

1. Give only when definitely indicated, give in adequate dosage, discontinue as soon as possible and resume treatment after a rest interval with great caution. These drugs have no place in the field of preventive medicine except under very extraordinary circumstances.

2. Keep the medication as simple as possible and prohibit sulfates, salines, high sulfur foods and pyramidon.

3. Maintain if possible normal urinary output with an alkaline urine, but not above p.h.7. This can be quickly and fairly accurately determined with nitrazene paper.

4. Administer sulfanilamide and sulfapyridine very carefully in diabetics, nephritis, anemias and those with preexisting hepatic damage.

5. If jaundice, febrile reactions, neuritis, skin reactions, delirium, stupor or enlarged tender liver occur, while administering these drugs, proceed with care.

6. Examine the urine frequently and if red blood cells, albumin, casts or crystals of acetylsulfanilamide or acetylsulfpyridine appear continue only with extreme watchfulness.

7. If more than two grains of sulfanilamide per pound weight in adults or more than three grains per pound weight in children have been given, do frequent white blood counts and hemoglobin determinations and if the hemoglobin drops rapidly or the total white count drops, especially with a relative decrease in polymorphonuclear cells, proceed with extreme caution.

8. The same rule applies to sulfapyridine if more than one and one-half grains per pound weight in adults or more than two grains per pound weight in children have been administered.

9. Dispense or prescribe only with proper warning and instructions, and only for the immediate need of the patient.

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TUBERCULOSIS: HOME TREATMENT

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In talking to you on the subject of home treatment of tuberculosis I am afraid I will get into very deep argument with many of the physicians of today. However, I am thoroughly convinced that we can do something for these patients in the home and that it should be done.

There are not enough beds to begin to think about placing all of these tubercular patients in a sanitarium. In 1931 Chicago had 1,125 beds for patients having tuberculosis. The beds were full. They had a waiting list of 7000. There were 3,300 open cases on the register. What are we going to do under such circumstances? There is no place to put them. There is only one solution—treat them in the home. Much good can be done, is being done and will continue to be done.

How I do my work is as follows: First I convince my patients with all the power that I have that they are tubercular, and until I convince them on that subject so that they are perfectly satisfied that they are victims of tuberculosis, I do nothing more. After they are well satisfied then we are ready to proceed.

This seemingly takes all the props out from under them. I next proceed to encourage them and build up hopes of future betterment. I sit down beside them and tell them that they can get well if they are willing to pay the bill—not financial altogether, but in performance of those things that they must do if they want to get well.

I try to teach them that there is only one cure for tuberculosis; that the treatment consists of rest, more rest and then rest some more. I also teach them that drugs are of no avail, and to center their hopes upon rest, plenty of good food and plenty of fresh air. In summer the windows should be wire open, and in winter the air should be warmed; there should be a certain amount of draft through the room all the time.

Visitors should be excluded except during such hours as I dictate to them. In patients who are very ill no visiting is allowed; others are allowed but a few visitors. As the patient improves, of course, they can have more visitors. But no one should come in and argue with the patient about how to get well. If a visitor begins to talk to the patient of other methods of therapy, they are immediately excluded from visitation.

The patient should be placed in a horizontal position with just enough pillows under his head to make him comfortable. From 9:00 to 11:30 A. M., the doors should be closed and no one, not even members of the family, should visit him. Also from 2:00 to 4:30 P. M., there should be no visitors.

I allow them to eat anything they like and when they wish. I impress upon them the necessity of pure wholesome food.

After keeping them in bed for six or eight weeks, I begin pneumothorax in the mild cases if they have not improved. I know that some specialists never would say to give a pneumothorax except in a hospital. I don't agree with them. I think it can be done safely in the home and I know that you can't get all of these patients to go to a hospital. The beds aren't there. Are you going to step aside and let these people perish just because you would rather do it in a hospital? I say no! We physicians have to work with the material we find and under the circumstances in which the patient lives. I have no fear at all of giving a pneumothorax in the home.

My method of giving a pneumothorax is as follows: I generally give from about 250 to 300 cc. of air at first. The second day I go back and give from about 300 to 400 cc., and then the third day I give them about 450 to 500 cc. according, of course, to the pressure reading on the monometer. The fourth day I give them another pneumothorax and my readings on the monometer tell me how much to give them. After this I give one every week for some time. And, of course, as the patient improves we give them farther apart—ten days, two weeks, three weeks, and sometimes even four weeks apart. Some of my patients only require 300 cc. of air while others require 900 cc. per week. Why? I do not know. But the improvement in the collapse therapy is wonderful. Many of the cases seemingly are beyond help, and still under the collapse therapy their improvement is wonderful.

Now as to exercise: When my patients are clear of fever for two months I begin to allow

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them to be up for a period of five minutes per day, and adding to that until they are up long enough to go to the table. That is generally about 30 minutes. In this way they get to eat one meal each day at the table. I do this because these patients should be happy, and there is no time as pleasant as the moments spent around the family table. The temperature is being watched all this time. If there is any rise in temperature there must be some explanation before they are allowed to be up again. After they are up about one hour I continue this plan for a couple of weeks. Then I lengthen the time, always clinging to the five-minute per day advance. When these patients are able to remain up four hours a day they are allowed to come to the office to get the treatment, returning home immediately while counting the time to and from the office as part of this four-hour period.

If the physicians of this part of the state will begin to look at tuberculosis as I look at it I think they will agree that we have been very negligent in our care of the tubercular, and that it is high time we should start caring for these patients ourselves or turn them over to someone else who will care for them. I know that the sanitarium is the ideal place but we can't all go to hospitals because our hospitals are full and there are not enough beds. Therefore, it is up to us to do the next best thing—that is, treatment in the home under the care of someone who will spend some time studying tuberculosis and its problems.

SOME POINTS ON OBSTETRICS AND EARLY PEDIATRICS IN GENERAL PRACTICE

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We still do not see our obstetrical patients early enough or often enough. Quite true, many need only watching, but others need treatment; and you can't tell which ones need treatment unless you see and examine all of them early and regularly.

The first time you see your patient get an essential history, brief, and to the point. Check up on past illnesses, especially scarlet fever, sore throat, rheumatic fever, and kidney trouble. Get the family history for kidney trouble, cancer, diabetes, multiple pregnancy and obstetrical history.

In the examination, look to the general conformation of the patient. The short, stubby or blunt-fingered patient—beware of her; the tapering, light-boned or feminine type generally does well. Look for thyroid, heart, lung, kidney, and liver pathology. Do a careful pelvic examination—external measurement and palpation, internal measurement of the anteroposterior diameter of the inlet, angle of the pubic arch, transverse diameter of the outlet, ischial spines and coccyx. I use calipers outside, but measure inside with my hand: if the full length of the left index finger fits between the symphysis and the promontory of the sacrum, the inlet measures nine centimeters anteroposterior; and if the fist fits, with the back of the hand along one ramus and the thumb-side of the first finger along the other ramus, the angle of the pelvic arch is ninety degrees plus. If the fist fits with the first and fourth knuckles between the ischial tuberosities, the transverse measurement of the outlet is nine centimeters plus. If the ischial spines are smooth and do not protrude, and the coccyx is freely movable, the indication is that a normal baby will come through in a normal delivery.

A blood count is helpful, a Wassermann or Kahn compulsory, and a urine analysis indispensable. Tell your patient your findings and your prognosis for labor, and warn her about danger signs such as headache, cramps, vomiting, spots before the eyes, dizziness and swelling of the feet. It assures the patient that you are taking care of her, and gives you a chance to avoid serious difficulties.

Adequate diet and exercise is essential. Control the weight gain to twenty or twenty-five pounds. Prophylactic treatment with iodine, cod liver oil, and calcium helps prevent thyroid trouble in mother and baby, bad teeth for mother, and insures adequate calcium metabolism in the fetus. I give Lugol's solution, drops ten, twice per week, or its equivalent, cod liver oil, U.S.P., drams one, t.i.d. or its equivalent, and one quart of milk daily or dicalcium phosphate, grains ten t.i.d. Zettelman has pointed out that vegetables alone are not adequate for calcium intake, and that at least one quart of milk or one-fourth

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pound of cheese or its equivalent is necessary for calcium metabolism.

Watch kidney function, especially during the last weeks of pregnancy. If the blood pressure is 140/90, look for trouble. See your patient every week of the last month of pregnancy. Don't let primiparas go over 280 days. Induce labor in primiparas after 280 days.

Don't hurry the labor after it has started. Use pituitrin, if at all, judiciously. Be sure the passage is adequate and the passenger in normal position. Use small doses, one to three minims at most; any more may give you a uterine tetany with disastrous results. Tell your patient that 18-20 hours is the average duration of labor for primiparas; that time is required to dilate the cervix and mold and expel the head. The average patient will understand and cooperate with you. Don't let grandma hurry you into trouble. I follow Falls who says not to interfere with labor as long as fetal and maternal hearts are all right, and pains are progressing normally with the fetus in a normal position. Tell your patient your rectal examination findings as you take them from time to time. It helps her to know how she is progressing, and assures her that you have the situation well in hand.

Avoid instrumentation and too much anesthesia. Use your chloroform or ether in the last of the second stage. I use chloroform in the home, in an ordinary drinking glass with cotton in it. Pour chloroform on the cotton and turn the glass over the patient's nose as the pain comes on. Have her take three deep breaths, hold the third, and push. The patient can give her own anesthetic this way, if necessary, and cannot get too much. Tell your primiparas how to push. They don't know how, or have been told wrong by some well-meaning soul. Stand ready to do an episiotomy when, and if, indicated by an impending tear. Have your tracheal catheter ready, and use it, if possible, before the babe takes a breath. This often avoids many difficulties in opening the air passages and getting the babe to breathe.

If repair is needed, close carefully with catgut, using a subcuticular stitch to close the skin. Examine the mother's pulse, uterine discharge and fundus. Before leaving, show her how to make sure that the uterus is contracted. Ergotrate, 1/320 grain, three to four times per day, is very helpful. Examine the babe carefully, especially tongue and extremities, also glands, liver and spleen.

Give the babe the benefit of breast feeding whenever and as long as possible. Watch the mother's nipples and breasts carefully. Use boric acid solution on the nipple before and after the baby nurses. If a mastitis appears use ice packs. For a central duct obstruction, Zettelman recommends heat. When a formula is necessary I use one of the dried or canned milks rather than pasteurized milk. Don't get the formula too strong or too sweet, and remember that all babes are different, or may be.

I think it is a good idea to circumcise all boy babies. I wait until he is gaining weight and has a fair start, somewhere between the third and fourth weeks. I use the clamp method, and find that most babes will go to sleep with the clamp on them. Set it tight, and retighten it as you can. Give it time. Let it set ten minutes at least.

Explain to parents that little medication is necessary. A tiny baby doesn't need castor oil for a cold on its bowel; nor does it need teas to break out the hives. Most colds in tiny babies are not colds—just dust and other irritants. If you can get the family to let the babe lie, stay away from it with upper respiratory infections, and do not pick it up every time it cries, you are giving it a chance for a good start in life. Brenneman uses as little medication as possible. If you can keep mother, grandmother, and the neighbors from using too many home remedies, it will be a great help.

I start cod liver oil, a concentrated form, at two months, with orange and tomato juice alternately, if tolerated; cereal at four months, with fruit juices and soups, gradually working in other soft foods until the babe is weaned from the bottle at about seven months. I gradually substitute pasteurized milk for a formula until the babe is getting straight pasteurized milk any time after seven months.

I have mother and babe come in for a check-up about six to eight weeks post partum. At that time I give the mother full information on contraceptive methods, and if possible, fit the patient at that time with an occlusive diaphragm, and teach her the technique of its use with a spermatocidal jelly. At the same time, I suggest that the babe be immunized for whooping cough,

diphtheria, smallpox and typhoid by the time he is one year of age.

SUMMARY

1. Examine all obstetrical patients early and often.
2. Tell your patient how she is, and your prognosis for labor.
3. Don't let primiparas go overtime.
4. Explain what you find, what you expect, and what is happening as it occurs during labor, and you have a cooperative patient.
5. Don't hurry your deliveries. Avoid use of instruments and too much anesthesia.
6. Examine mother and babe post partum.
7. Start babes right and they'll have a better chance.
8. Give as little medication as possible. Use preventive medicine.
9. At six to eight weeks post partum reexamine mother and babe, and give contraceptive instruction at that time.

PERITONSILLAR EDEMA IN DIPHTHERIA. DANGER OF INCISION

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Since much has been written in the literature concerning the disease "diphtheria," a lengthy discussion of its etiology, symptoms, diagnosis, prognosis and treatment should, therefore, not be necessary. Yet the mortality of peritonsillar edema of diphtheria in adults remains surprisingly high, due perhaps to the fact that it is often thought to be a peritonsillar abscess. In many cases the membrane is visible only on careful examination of the posterior surface of the uvula and pharynx, and then considered as a non-diphtheritic exudate. With the development, however, of peritonsillitis, redness and edema of the faucial pillars, soft palate and uvula, the clinical diphtheria in adults is easily mistaken for peritonsillar abscess. Incision of the peritonsillar space is performed. There follows a marked production of membrane which within twelve to twenty-four hours covers uvula, soft palate, tonsils, pharynx, nasopharynx, etc. The swelling of the neck is so marked that it interferes with respiration. Extreme toxemia is present.

From the Department of Otolaryngology, Christie Clinic.

In the differential diagnosis between the two infections the following are more characteristic of peritonsillar edema of diphtheria than of peritonsillar abscess:

1. No history of previous sore throat accompanied by high fever and chills is necessarily present.
2. There is uniform concurrent involvement of both areas, that is—both sides equally involved, not one preceding the other as in abscess.
3. Glands in the neck are usually bilateral, not tender and not discrete.
4. Hyperemia is uncommon.
5. Toxicity is notably marked.
6. Local fluctuation is absent.
7. Marked pain in throat is infrequent.

It is the repeated occurrence of incision in these peritonsillar edema cases which warrants our careful attention. A typical example is presented in the following case:

Mrs. W. S., twenty-three years of age, consulted her family physician on February 12, 1939, with the complaint of having had an attack of influenza with mild sore throat for one week. On examination he found "enlarged and tender tonsils with abscess formation." An incision was made into each peritonsillar swollen area, but no pus was obtained. This procedure was repeated the next day with still negative results. The following day her condition became alarmingly worse, and on admission to the hospital she was breathing laboriously and coughing frequently. The patient had difficulty in opening her mouth; tonsils and surrounding tissue were considerably swollen, tender and covered with a white membrane. A smear and culture of the throat was taken. The axillary temperature was 101.8 F., pulse 104, respirations 22. Erythrocytes numbered 4,300,000 and leukocytes 26,100 per cubic millimeter of blood. The differential study of the stained blood smear revealed 88 per cent. neutrophiles, 11 per cent. lymphocytes and one per cent. large monocytes. Urine showed three plus albumin, one plus acetone and five to ten pus cells, but otherwise was negative. The direct smear from the throat showed cocci of the streptococcus and pneumococcus type and few scattered gram-positive rods. A smear from the culture taken only four hours later, revealed an increase in the gram-positive rods, several of which had a morphology resembling the diphtheria bacillus. The report of the eighteen-hour culture was positive for Bacillus diphtheria. With the final report she was given 40,000 units of diphtheria antitoxin. In twelve hours this was repeated 1,000 cc. of five per cent, glucose in normal saline given and oxygen per nasal catheter started.

Patient's condition gradually became worse, and consultation was sought. I saw the patient for the first time at four o'clock in the morning of February 16, 1939. At that time the patient was irrational, very

restless—rolling and tossing in bed, cyanotic, dyspneic as represented by supra- and infra-sternal retraction, skin dehydrated, axillary temperature 103.6 F. Pulse weak and rapid—recorded 144; respiration 30. A greyish, dark membrane was present over both tonsils with the surrounding tissue so edematous that the pharynx could not be seen. Bilateral cervical glands were palpable. The patient was a true picture of malignant diphtheria.

Under local anesthesia a low tracheotomy was immediately performed. Upon excising a portion of a tracheal disc, the inner wall of the trachea was exposed and observed. Completely covering the mucous membrane was a greyish, white fibrous-like membrane. By means of a long narrow tissue forcep, a firm hold on the membrane was obtained, and with gentle pull was removed, apparently in its entirety, from the trachea and bronchi (see photograph). With the exterpation of the membrane the patient's respiration ceased being labored, restlessness soon vanished, and her rationality returned. She was placed in a large steam tent, an ampule of coramine given and intravenous glucose be-

Figure 1. Tracheobronchial diphtheritic membrane.

gun. The would-be excellent results were, however, short lived, for some six hours later the patient's axillary temperature suddenly jumped to 105.6 F., pulse was recorded around 168 with respirations of 40. Heart tones were distant and exodus occurred soon after. Final or terminal diagnosis was toxic myocarditis.

The high fatality noted in these cases results not so much from the delay in the administration of the antitoxin as from the increased rapidity of absorption of the diphtheria toxin. The extension of infection is encouraged by the trauma produced with the incision. Gordon and Young,[1] as well as Neffson and Brem,[2] in their series, noted that if the error of incision is remedied by the administration of diphtheria antitoxin within twenty-four hours, no appreciable harmful effect was observed. When the antitoxin was given later than twenty-four hours after incision, the mortality was more than doubled. The percentage of fatality was far less, however, when the antitoxin was given shortly after incision, but increased rapidly thereafter when delayed twenty-four hours.

Tracheotomy or intubation[3]—which procedure is desirable? Cyanosis and dyspnea as represented by supra- and infra-sternal retraction are external evidence of obstruction high in the respiratory tract. Cyanosis of rather sudden laryngeal obstruction is unmistakable, and emergency measures are immediately undertaken to give relief to the patient. Whereas restlessness, cyanosis, high fever, dehydration, rapid and weak pulse so consistent with obstruction of bronchioles are not ominous and are consequently not identified as obstructive symptoms, they are more often associated with the toxemia.

These infections of diphtheria in which peritonsillar incisions have been made are usually of a descending type. The symptoms of obstruction are gradual and undramatic, and unless they are being watched for may not be recognized. Low tracheotomy appears to be the method of choice, in view of the descending nature of the infection. By this surgical procedure the inner surface of the trachea can readily be seen and studied, and the treatment of the tracheobronchitis can best be handled. As in the case presented, the membrane was removable and instillation and suction easily applied.

IN CONCLUSION

Diphtheria is usually considered a disease of childhood.[4] When present in adults, and then usually associated with peritonsillar edema, it is easily mistaken for peritonsillar abscess. The tendency to incise is great with extremely high fatality inevitable. This fatality results from the increased rapidity of absorption of the diphtheria toxin encouraged by the trauma produced with the incision. Gordon and Young recommend the immediate administration of diph-

theria antitoxin whenever a supposed peritonsillar abscess has been incised without the delivery of pus. Any delay longer than twenty-four hours in the administration of the antitoxin invariably leads to death. Because of the descending nature of the infection, low tracheotomy appears the method of choice for relief of respiratory obstruction. Rarely, if ever, is the incision of a peritonsillar abscess so urgent that it cannot be delayed until the report of the culture has been obtained. Hasty incisions may be regretful, delays can do no harm and may help to prevent and diminish the prolonged morbidity and high mortality in these cases.

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TRACHOMA AND FOLLICULAR CONJUNCTIVITIS

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It is rather unfortunate that the terms "trachoma" and "granulated lids" are often used as one and the same. Granules in the palpebral conjunctiva are one of the most important findings in trachoma and it is natural for the examiner to look for this disease whenever they are present. However, the appearance of granules or follicles in the palpebral conjunctiva is not limited to trachoma alone; therefore, the term "granulated lids" does not designate a definite diagnosis. Besides trachoma, follicles may be found in swimming pool conjunctivitis, follliculosis, chronic follicular conjunctivitis, toxic conjunctivitis, and, in rare cases, in simple acute conjunctivitis. Parinaud's conjunctivitis, characterized by yellowish granular, sometimes ulcerating, vegetations in the conjunctiva, and by a swelling of the preauricular gland, may also take the appearance of "granulated lids" in the beginning. The course of this disease, however, will soon decide the diagnosis.

It is not surprising that inflammations of the conjunctiva in which follicles are found are often confused. A follicle in itself does not show any characteristic feature, but is rather an anatomical reaction of the conjunctival tissue which can be brought on by different causes (Morax). It is the reaction of the tissue surrounding the follicle which, together with the information gained by the history of the case, the course of the disease and the corneal findings, renders a differential diagnosis possible. Due to the fact that there is an endemic occurrence of follicular catarrh and folliculosis in the southernmost part of Illinois, together with the occurrence of true trachoma in the same district, we had the opportunity to study the clinical aspects of these diseases, and to compare them with each other. Swimming pool conjunctivitis has not been seen in any of our clinics and is not to be dealt with in this paper. It may be mentioned that it appears under the picture of an acute follicular inflammation of the conjunctiva and resembles trachoma for a short time. However, it will clear up in about a month, never leads to corneal complications and does not leave any traces (Morax).

FOLLICULOSIS is a disease of the childhood and is often discovered in children when they are examined in school. It presents the formation of follicles in the conjunctiva in its purest form, for in this disease there is no other pathology found. There are no inflammatory symptoms whatsoever. No epiphora, no photophobia, not even a burning sensation give rise to complaints. The children are not aware of the fact that there is anything abnormal in their eyes and one is often surprised at the high degree of folliculosis of the conjunctiva palpebrae and fornicis that may exist without giving rise to any symptoms. In some children there are only a few glassy follicles in the lower fornix. In others, the formation may be so extensive that the lids appear slightly bulging, and at eversion of the lids a regular "package" of follicles is to be seen protruding from the conjunctiva palpebralis. These follicles often invade (contrary to trachoma) the conjunctiva bulbi in the fornix. They are roundish or egg-shaped, of pinkish white color, and Morax compares their arrangement to that of a rosary. In other words, each follicle is distinct and, due to lack of infiltration of the neighboring tissue, well defined. The conjunctival epithelium is undisturbed. There is no edema, no

From the Governor Horner Southern Illinois Trachoma Clinics. Med. Director, Dr. Harry S. Gradle.

hypertrophy, no velvety appearance. The conjunctiva of the tarsus has its normal transparency, the meibomian glands are clearly seen and the conjunctival vessels are undisturbed. The follicles are mostly in the fornices and only a very few small ones can be detected in the tarsal conjunctiva of the upper lid. The follicles never burst spontaneously. On expression with a roller forceps a gelatinous greyish white material is seen. Inclusion bodies were never found in this material nor in simple epithelial smears (Dr. Thygeson of Columbia University kindly examined the smears for us). Lindner states that he examined over a hundred similar smears and found no sign of inclusion bodies. Morax transplanted expression material into five human conjunctivas with completely negative result. Folliculosis does not lead to corneal complications and clears up without treatment in two to four years, leaving no traces.

FOLLICULAR CATARRH has somewhat smaller follicles than are found in folliculosis. Each follicle is clearly defined, but in addition there is a slight catarrhal reaction of the rest of the conjunctiva. This reaction is never strong enough to lead to a severe hyperemia nor to edema of the conjunctival tissue. The conjunctival vessels and the meibomian glands can always be recognized. The catarrh may give rise to a few subjective symptoms such as burning and itching, especially in the morning. However, it is rarely that one finds marked secretion from the conjunctiva. If it is present it is more watery than mucopurulent. The condition has always a chronic character and never leads to photophobia, blepharospasm, nor diminution of sight. Follicular catarrh may resemble the mild forms of beginning trachoma. Trachoma, however, will soon give rise to more marked subjective symptoms. Further, at a certain stage the trachomatous follicle has a tendency to burst spontaneously, or after slight massage—a finding which is never encountered in follicular catarrh. Corneal complications are said to be unknown in fallicular catarrh. The most extensive formation of follicles may exist for years without damage to the cornea. Only once did I have the opportunity to observe a seven-year-old boy who developed a superficial punctate keratitis in a case which I believe was follicular catarrh. There was no vascularization of the cornea, and the superficial infiltrates, which perhaps were caused by mechanical rubbing of the follicles on the cornea, healed within a week under treatment with White's ointment, Oxycyanide eye wash and vitamins A and D. The follicles persisted in this case after the disappearance of the corneal findings, and in order to avoid a similar exacerbation were expressed with a roller forceps. No recurrence has appeared. I mention this instance to show the difficulties one may encounter in trying to diagnose these cases correctly. Some cases require more than one examination for the differential diagnosis.

Follicular catarrh is an infectious disease and is probably caused by the Bacillus granulosis Noguchi, a small gram-negative bacillus (Lindner). The disease apparently spreads easily among children and adolescents. Its course is from one to three years and it clears up without leaving any scars.

As I mentioned above, beginning trachoma may resemble follicular catarrh. Very soon, however, the palpebral conjunctiva will change its appearance and the examiner will know which disease he is dealing with. Trachoma is a formation of lymphocytic granules in the conjunctiva, *plus* a chronic (or sometimes even an acute) reaction of the rest of the conjunctival tissue leading to an infiltration of the whole mucous membrane, including the underlying tarsal cartilage. The conjunctiva will look edematous and have a more or less muddy appearance. The follicles, which usually are deeply imbedded in this hyperemic edematous conjunctiva, are more confluent with the surrounding tissue than in other follicular diseases. The conjunctiva as a whole has a thickened, velvety appearance. The existing hyperemia extinguishes the finding of the blood vessels which are normally seen over the tarsal plate. The same is true for the normally visible meibomian glands. Due to the loss of the transparency of the conjunctiva these structures become invisible. It has already been mentioned that the follicles of trachoma have the tendency to burst, although it may take several weeks of treatment to effect this. Scar tisue will develop after the bursting and is easy to recognize due to its whitish uniform color. Scars in the conjunctiva are, however, a sign of a later stage of trachoma, and if they are present there will be no doubt as to the character of the dis-

ease, as the formation of scar tissue occurs only in trachoma.

Having these differential points in mind, it still might be difficult to make a definite diagnosis in some cases. Morax states that simple folliculosis should never be confused with trachoma. Follicular catarrh, however, should, in our opinion, be looked at as "suspect trachoma" for a period of several months, because it might be possible that one is dealing with a beginning case of trachoma. This is especially true in areas where trachoma is prevalent, and one neglected positive trachoma case may be the source of infection for many others. It is, however, not necessary to keep the patient under this tentative diagnosis for a long period, as the course of the disease will soon lead to a diagnosis. MacCallan, who gained wide experience in the trachoma work of Egypt, states emphatically that even in the earliest stages of trachoma one can always find the beginning ingrowth of blood vessels between Bowman's membrane and the corneal epithelium combined with small subepithelial cellular (lymphocytic) infiltrations at the end of these vessels. It is possible that the examiner needs a corneal microscope to see the trachomatous pannus in the very early stage, but within a few months pannus will become distinguishable with focal illumination only. A case of follicular catarrh never develops such changes. The more careful one looks for pannus the sooner a positive diagnosis of trachoma can be made with certainty. Morax is certainly right when he states that the physician should not call a disease trachoma until he is certain that this disease is present. Trachoma is too serious a condition, and its consequences too frightful to mention such a diagnosis without absolute certainty.

The question arises: are folliculosis, follicular catarrh and trachoma related to each other? Particularly in a district where all these diseases are relatively frequent and endemic, as is the case in southern Illinois, this question becomes a very acute one. Although the literature on the subject now states rather uniformly that we are dealing with different conditions, one is forced to raise this question again and again. This is especially true in families where the children show a definite follicular catarrh, or perhaps only a folliculosis for many years, while the father or mother have a well-established case of trachoma, usually with deep scarring of the conjunctiva and extensive pannus. It may be possible that these children become superimposed with the trachomatous infection. Lindner mentions instances in which soldiers who had follicular catarrh during the world war and were wrongly diagnosed as having trachoma, actually got trachoma after they were mistakenly isolated with true trachomatous patients. We believe that the children in our district contract the follicular catarrh in school, and that the finding of trachoma in the same family is merely a coincidence.

The character of folliculosis as an infection has been denied, especially by Morax whose above-mentioned experiments to transmit pure folliculosis were negative. Follicular catarrh on the other hand has been established as an infectious disease, especially by the well known self-experiment of Axenfeld who inoculated himself and developed a follicular catarrh which cleared up after one and a half years. If many cases of pure folliculosis are seen within a small region, as we find in three counties of southern Illinois, then it is hard to deny the infectious character of this disease, and to explain it, as it is done in the literature (Koopmann—quoted by Schieck) simply as due to a lymphatic constitution. Schieck indeed does not differentiate between folliculosis and follicular catarrh and describes both conditions under the heading "Follicular Conjunctivitis." Poor nourishment might possibly be a factor in bringing about folliculosis. Over a period of several months we gave several of our patients large doses of vitamins but could find no beneficial influence of this medication. Morax himself states that we do not yet have a satisfactory explanation for the cause of folliculosis. Whatever the case may be, we are sure that folliculosis and follicular catarrh are harmless conditions, usually found in children and young people, while trachoma is a severe eye disease which carries all the dangers of corneal complications from simple vascularization to ulceration and scarring of the cornea with resulting blindness.

It might be well to keep every case of folliculosis and follicular catarrh under regular observation to exclude any possibility of trachoma. An antiseptic eye wash should be prescribed in these cases, and the patient should be instructed as to the necessary hygienic precautions (separate wash water and towel). However, strict measures such as surgical interference or isolation do not seem to be indicated in these relatively harmless conditions. It is true that an expression of

the follicles, as it is practiced in true trachoma, hastens the recovery in folliculosis and follicular catarrh, and the course of the disease is frequently shortened to one or two months, especially if the expression is followed up by one per cent silver nitrate treatment twice a week for one month. Massage with chaulmoogra oil or an antiseptic solution also helps toward a quicker recovery. It is, however, a matter of subjective preference if one wishes to employ these measures in non-trachomatous follicular diseases which show practically no symptoms, are not dangerous and clear up by less drastic means.

Sulfanilamide, which has a very beneficial effect in fresh cases of trachoma, does not alter the course of a simple folliculosis or follicular conjunctivities. We gave Sulfanilamide to several children with these afflictions and found no perceptible change. This seems to be another proof that we are dealing with conditions which have no relation to true trachoma.

SUMMARY

Findings of formation of follicles in the conjunctiva do not always indicate the presence of trachoma. Many children in Southern Illinois are affected with folliculosis and follicular catarrh, conditions which are relatively harmless and do not require rigid measures. Such cases should be kept under medical observation, in order to exclude the possibility of true trachoma. It is not always possible to make the differential diagnosis at the first visit. However, certain definite characteristics always develop in true trachoma, without which one should not make a positive diagnosis.

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AN EYE FOR A TOOTH OR A TOOTH FOR AN EYE

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Though isolated instances of eye disease associated with dental infection were reported in the past century, the modern interest in the subject dates from the comprehensive article by William Lang in 1913 on "The influence of chronic sepsis on eye disease" (Lancet I p. 1368). The subject was further advanced by Billing's work in 1916, in which focal infection was studied from a more general viewpoint. In 1928, Russell M. Hayden contributed his important monograph on "Dental Infection and Systemic Disease," in which the previous clinical and experimental work is summarized and elaborated.

Clinical reports indicate that dental disease may induce dysfunction of the visual apparatus in almost any part, either directly or indirectly. One of the earliest cases to create interest was reported by Bier in 1817, where a contraction of the visual field was alleviated by removal of a carious tooth. However, the eye conditions associated with a dental focus of infection are in order of frequency, first, diseases of the uvea—iritis, iridocyclitis, choroiditis, and chorioretinitis; second, involvement of the cornea—infiltration or ulcer; third, conjunctivitis, scleritis and episcleritis; fourth, edema of the lids and blepharitis, papillitis, and retrobulbar neuritis; and fifth, functional disturbances.

The interrelation of the eye and focal infection is probably better capable of evaluation than similar pathological reactions elsewhere, since the patient with an ocular disease comes for treatment early, the course of the malady is readily followed, and the reaction to treatment is comparatively rapid. Whether a focal infection will affect the eye or not depends on the specific virulence of the organism, the amount of toxic absorption, and the local and systemic resistance of the patient.

Experienced ophthalmologists have estimated that about 40 per cent. of cases involving the uvea are due to focal infection, and that about one-third of this group is of dental origin. In young individuals the tonsils are a common factor, but past middle age dental sepsis is probably more frequently responsible. It is oftentimes difficult to decide whether an eye condition is due to an ordinary focus of infection or secondary to a focus of tuberculosis. Acute conditions are more likely to belong to the former group. In chronic disorders tuberculosis must be suspected, but inasmuch as dental infection can be easily eliminated, one should first attend to the teeth in a dubious case. In a recent report of 52 eye cases suspected as originating from focal infec-

tion, 21 or 40 per cent. were alleviated by elimination of suspected teeth.

There are four possible ways in which the eyes may be affected by diseased teeth. First, continuity. Involvement of the soft tissues of the cheek or the underlying periosteum may extend to the orbit or, more rarely, the orbit may be involved through a maxillary sinusitis of dental origin. Thrombophlebitis in this region may end in acute or chronic cavernous sinus thrombosis. Second, extension along nerve sheaths. Herpes of the cornea may thus occur through involvement of the Gasserian ganglion or following the connecting filaments of the second and first branch of the ophthalmic nerve. Third, reflex. Ocular symptoms may accompany irritation due to impacted or ectopic teeth and pulp stones or pulpitis and so cause an otherwise unexplained neuralgic pain in and about the eye. Fourth, and most important, metastasis of infection through the lymphatic and arterial circulation. Eye involvement may thus arise spontaneously, or be precipitated by debilitating conditions, such as injury to the eye, anemia, diabetes, tuberculosis, or syphilis. Ocular involvement occasionally, though rarely, follows dental extraction. Traut was able to obtain a positive blood culture in acute iritis which was identical with the organism cultured from the focus of infection.

Focal infection, transmitted by the circulation, is generally due to various strains of streptococci. Their elective localization is probably due to acquired organ specificity. Haden discovered streptococcus viridans in 92½ per cent. of 1,500 periapical infections. Rosenow found that two-thirds of the guinea pigs injected intravenously with cultures of dental granulomata from patients having eye diseases exhibited metastatic eye infections, while in the control group only 14 per cent. became thus affected. He has also shown that the migration rate of streptococci in an electric field varies with their organ tropism. However, since exposure to a high frequency current changes their characteristics in this respect, it is not improbable that the properties involved in specific localization are acquired phenomena.

Focal infection implies metastasis of bacteria or toxins, but it is not a necessary sequel to every focus of infection. Evidence of present or past dental caries is present in three-fourths of the adult population. Investigation has demonstrated that infection involves the pulp of every tooth in which the dentine has been invaded by caries, whether the tooth is vital or not. The tubular nature of dentine presents very favorable conditions for pulp invasion, and hence infection may even occur without pulp exposure. When the pulp is dead, or moribund, the tooth is called "pulpless," and bacteriological tests have shown nearly all pulpless teeth to be infected. That focal infection does not occur in all who have the potential possibility is due to the local and systemic resistance of the individual.

In the presence of focal infection, the dental conditions listed below should be considered as having a possible etiologic relationship in the following order.

(1) Devitalized teeth. The most dangerous are those that show on an x-ray a non-granulomatous rarefying osteitis, that is a periapical diffuse dark area without definite limitations, which indicates that the infection is not well walled off. Those with "good" root fillings are most likely to show such areas. Thickening of the periodental membrane is another significant sign.

(2) Stumps, just below the mucous membrane. These should always be removed regardless of x-ray findings, as they are always badly infected.

(3) Devitalized teeth with granulomata. The granuloma is a defensive mechanism which occurs mostly with those teeth that either have no root fillings or only partially filled root canals. It may or may not show on the x-ray film.

(4) Devitalized, x-ray negative teeth. Such teeth may be even more dangerous than x-ray positive teeth, as the lack of x-ray findings indicates less resistance to infection.

(5) Tooth remnants surrounded by alveolar bone. Edentulous mouths should be x-rayed as otherwise these root remnants would be overlooked.

(6) Residual infection in alveolar bone.

(7) Pyorrhea alveolaris.

(8) Acute apical abscess (acute suppurative periodontitis).

(9) Impacted unerupted teeth. The tubular nature of dentine presents favorable conditions for pulp infection which may occur even without pulp exposure.

(10) Degenerating vital pulps. Hidden caries should be sought and all fillings scrutinized.

Since most of these dental conditions are free from local symptoms, they are generally unsuspected. The infection being in osseous tissue does not allow of localized expansion and drains perforce into the lymph and blood. Haden found that three-fourths of the eye cases associated with dental sepsis were due to periapical infection. The patients suffering from pyorrhea, however, were in surprisingly good health owing to the open drainage in this condition. But periapical infection is constantly subject to the pressure of mastication, as each bite has the force of seventy pounds to the square inch.

The prophylaxis of dental sepsis involves the prophylaxis of dental caries which is the ultimate cause of pulp infections. In vital teeth, prompt and adequate care of the primary caries will generally terminate the secondary pulp infection. The sooner the dental foci are eliminated after the onset of any disease, the better are the immediate and end-results. After the focal source has been removed, a marked lessening of inflammation is observed, generally in forty-eight hours. If not, a further search must be made. It is poor practice to curet infective alveoli after extraction, as one thus destroys a defensive barrier built by nature. The safer procedure is to let nature throw off the granuloma in her own manner. If an eye disease responds rapidly to local treatment, the removal of the suspected focus should be delayed until the eye has recovered. When the patient's general condition permits, all dental foci should be removed at one operation. Otherwise, one or two infected teeth may first be removed, and an autogenous vaccine administered to raise resistance before the removal of the rest. Vaccines will probably act more favorably, though, after all the septic foci have been removed. Few complications, local or systemic, follow removal of dental foci because of the low virulence of the streptococci present. Before undertaking an intraocular operation, infected teeth should be removed some weeks previously to avoid the possibility of postoperative uveitis from this source.

In the management of eye disease associated with dental sepsis, conservatism may be too costly. If we must choose between a tooth and an eye, the saving of sight is the primary consideration. Artificial teeth function, but not artificial eyes.

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THE MANAGEMENT OF GASTRO-INTESTINAL DISEASE IN PULMONARY TUBERCULOSIS

(An Internist Looks at the Treatment of Pulmonary Tuberculosis 11)

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The prominence of gastro-intestinal symptoms and complications in the diagnosis, prognosis and treatment of tuberculosis of the lungs warrants far more careful attention and study than it usually receives.

As I have said[1] the specialist tends to overlook or ignore new or even standardized procedures of proven efficiency in other branches of medicine however closely related to his own field. This attitude reflects itself in institutions as sanitoria dedicated to the treatment of certain aspects of a particular disease. As a result such institutions may be far less successful in the management of the complications of their particular kind of illness than is justified by the existing fund of medical resourcefulness. The state of our medical knowledge, in other words, may justify far more efficient treatment of complications and incidental illnesses than is usual in institutions devoted to the treatment of a single disease in a special organ. More directly such lack of acquaintance with the facilities of neighboring medical fields may be due to lack of interest or training in the medical personnel or a form of medical snobbishness, rather proud of its narrow specialization as characterized by entire ignorance of the everyday tools used with such good effect by colleagues in other branches. This attitude leaves such institutions unequipped mentally, and physically from the laboratory and diet kitchen standpoints to cope with anything except the problems incident to its specialty in the narrowest sense.

It is regrettable that the splendid progress in the treatment of pulmonary tuberculosis has not been paralleled by a similar successful management of its important gastro-intestinal manifestations. The ready availability of methods advantageously employed in a neighboring field of internal medicine and their neglect in tuberculosis may be explained by the extreme concentration of interest and time in solving the urgent problems of the primary thoracic disease.

On the other hand the gastro-intestinal expert may err by failing to recognize abdominal disten-

sion, gas, and frequent stools or constipation as the symptoms of pulmonary tuberculosis. The gastro-intestinal symptoms may long antedate the pulmonary complaints. Abdominal disturbance may be the only expression of disease in the lungs.

Bowel disturbances in tuberculosis are of two types: that with structural change and that with only abnormalities of function. Of the first type, tubercles followed by ulceration mark the classical form of tuberculosis enteritis. Tubercles and ulcers may occur from the lips to the anus. Goldberg, Sweany and Brown[2] found ulcers most commonly in the cecum. The ileum was the next most frequent site, with the colon a close third.

The tubercles are generally assumed to be due to the direct invasion of tubercle bacilli from the bowel lumen. Experimentally intestinal lesions have been produced by feeding the bacilli, but only in a modest proportion of the attempts. By first producing a deficiency of vitamin C, McConkey[2] succeeded in producing intestinal lesions in 26 of 37 guinea pigs inoculated by the oral route. In the human the usual manner of ingesting tubercle bacilli would be by the universal practice of swallowing sputum. In such instances the bowel tuberculosis is secondary to the pulmonary invasion. Of course, children may acquire their primary infection via the intestinal tract. The bovine strain is so introduced. The swallowed pyogenic bacteria may of themselves set up an inflammation in the gastro-intestinal tract. This nontuberculous inflammation favors the penetration of the tubercle bacilli according to some.

The sputum may not be the only means of introducing tubercle bacilli into the digestive tract. Viable bacilli are said to be excreted in the saliva and bile. Some authorities consider the blood as the usual route of intestinal invasion. Calmette holds this view. Stenken and Petroff[2] found intestinal tubercles in 17 of 25 guinea pigs infected by injection into the left ventricle.

Gastro-intestinal disease may be due to poisons elaborated in distant tuberculous foci. Both organic and functional intestinal disease may be due to tuberculosis in other organs, without any tubercles in the digestive tract itself.

Amyloidosis is tissue change common in tuberculosis and in other chronic infections. The infected area may be in some remote part of the body, far from the amyloid deposit.

Tuberculous ulcers are often entirely symptomless and unsuspected. Diarrhea, the commonest symptom, is usually of the putrefactive type. It may alternate with constipation or be supplanted by it. The putrefaction is due to the common hypo- or achlorhydria with its resultant failure of gastric digestion of proteins, leaving such proteins, particularly of connective tisue, to be disintegrated by bacteria in the lower ileum and in the colon. Further contribution to the putrefying process is made by the products of the bowel wall itself. The ulcers exude a decomposing protein. The mucosa between the ulcers is usually irritated to a high degree and weeps large quantities of decomposable secretion. The swallowed sputum is already undergoing proteolysis.

Pain is almost always absent in uncomplicated tuberculous ulceration of the intestine. Its occurrence signifies complicating dyspepsia, stenosis or peritoneal involvement. It is sometimes noted in tuberculous appendicitis as discomfort or recurring distress in the right lower quadrant. Ulcers of the mouth, pharynx and esophagus may be very painful leading to marked dysphagia and consequent emaciation.

Distension and borborygmi are common complaints in ulceration of the intestine. Tympany is a common finding. Rigidity of the abdominal wall is characteristically lacking even in tuberculous appendicitis.

Anemia is often especially severe and progressive in tuberculosis of the gastro-intestinal tract. It can be attributed to several factors. Hypochlorhydria is said to interfere with the absorption of iron. Poisons elaborated by the tuberculosis and by putrefaction interfere with hematopoiesis. They have even been presumed to increase the destruction of the formed elements of the blood. The loss of protein in the diarrhea depletes the blood proteins.

With involvement of the colon, particularly if distal to the hepatic flexure, the findings will be those of ulcerative colitis: pus and blood in diarrheal stools. Provided other sources of blood such as oozing gums and swallowed bloody sputum can be eliminated, the finding of a positive benzidine test on a meat-free diet is the most delicate evidence of ulceration. If the ulcer is in the ileum or higher, pus and microscopic blood may be digested before the stool is evacuated. Ulcers may heal, frequently with stenosis. They perforate rarely. Anal fistulas are

common sequels of tuberulous ulcers of the rectum. The x-ray evidence of definite ulceration is limited to Stierlin's sign of filling defect in the cecum.

The second type of structural change, if not advanced, if usually symptomless. As a cause of diarrhea it may be unsuspected unless fat in the stools or albuminuria due to a parallel amyloidosis in the kidney calls attention to the possibility of amyloid in the intestinal mucosa, one of the sites of predilection for its early deposition. The intravenous injection of Congo Red will throw further light on the extent of the process.

Functional bowel disease may complicate or be independent of ulceration. Common in tuberculosis, it is one of the manifestations of the many disturbances in physiology due to the still obscure toxic substances produced by tuberculous inflammation. Under this heading come most of the common digestive complaints in the tuberculous. Certainly in the majority of patients x-ray gives no findings or shows only minor disturbances as hypertonicity, hyperperistalsis, spasm or more rarely atony. The complaints commonly exceed the x-ray and fluoroscopic signs of trouble. The functional disturbances in the intestinal tract of the tuberculous include the dyspepsias of Adolf Schmidt.[3] In contrast to the paucity of complaints in uncomplicated ulceration or tubercle, the dyspepsias produce marked symptoms. The criteria for their recognition and the procedures for their control are well established. In digestive disturbances uncomplicated by tuberculosis, Schmidt's method of analysis and treatment have long yielded the most consistently good results.

Tuberculous patients afflicted with functional gastro-intestinal disturbances are either primarily introspective, neurotic or "neuromuscularly hypertensive,"[4] or they become so after having described their obviously functional disturbances to a succession of doctors. The impatience of the physician, confronted by a number of other very acutely serious conditions, is usually thinly disguised. After an endless number of drugs and dietary experiments these patients either come to regard themselves at fault, or the condition as irremediable.

In the production of the functional types of intestinal disease, the dyspepsias of Schmidt, the elaboration of tuberculous toxic substances result in deficient gastric secretion of acid and in hyperperistalsis and spasm. The effect of such toxic substances upon secretion and absorption in the small and large bowels is largely unknown. It is also uncertain whether the tuberculous toxin acts through its effect upon the vegetative nervous system or by directly affecting the intestinal glands and musculature. The hypo- or achlorhydria so common in pulmonary tuberculosis can in itself give rise to gastrogenous diarrhea by allowing rapid expulsion of unemulsified food material from the stomach. Too little hydrochloric acid means a failure of the acid barrier between the outside world and the intestine. The benefit of hydrochloric acid in the stomach may not be the direct destructions of tubercle bacilli but one of the direct acid effects upon the swallowed pyogenic bacteria, the proteolytic substances of the sputum. The acid also induces a longer, more complete gastric digestion.

The procedures indicated for analyzing the digestive functions do not differ from those usually employed in uncomplicated gastro-intestinal disorders. They should include a painstaking history describing in detail the dietary habits before and since the development of clinical tuberculosis. The intake of the principal calorigenic and vitamin-containing foods should be known. Defecation habits and the use of laxatives or bran should be recorded. The amount of tobacco, coffee, tea, candy or such medicines as cod liver oil, tonic or cough medicines affecting digestion either directly or indirectly should be quantitatively determined. Not least important and frequently overlooked are the patient's likes, dislikes and food idiosyncrasies. Too frequently diets are prescribed at a chart desk or in the diet kitchen instead of at the patient's bedside and without consulting the one who has to eat them. The duration of the complaints, their continuity or intermittency and their tendency to progress or remit with their relations to the kind of food and to the time of eating and defecating should be noted. The patient should describe his stools. He should tell the result of previous investigations and attempts at relief.

The abdomen should be examined for distension, peristaltic waves, tumors or swellings, tenderness and fluid. After a careful examination of the anus the lower colon can be inspected

through the sigmoidoscope. Material from the colonic wall should be examined bacteriologically and for amebae. Distinguishing tubercle bacilli from the other acid-fast bacilli of the lower bowel is not easy but possible. With a positive sputum it is unnecessary. In doubtful cases injection of guinea pigs with ulcer scrapings or macerated biopsy material should be utilized more often.

It is necessary to learn the ability of the stomach to secrete acid and rennin under the stimulus of food. The simple Ewald meal will suffice. Testing under alcohol and histamine stimulation is not indicated.

All stools should be examined and the findings recorded. Routine examination of the stools is neither difficult nor time-consuming. It includes a description of the consistency of the stool and of its diameter, if formed, its reaction to litmus, the presence of gas, its putrefactive or fermentative odor, the presence of pus, mucus and gross or occult blood, and of starch, fat or undigested food particles.

In the fluoroscopic examination esophageal diverticula are carefully sought. Esophageal deformities are recognized as occasional complications of thoracic tuberculosis. The examiner should observe tendencies to hyperperistalsis and spasm, especially in the cardiac, pyloric and ileocecal regions. Tenderness can be anatomically located. By allowing evacuation of the barium enema, inflating the colon and taking films, the "mucosal pattern," ulcers and colonic diverticula can be visualized. Ulcers between the duodenum and the terminal ileum cannot usually be demonstrated.

Putrefactive dyspepsia or its aggravated form, putrefactive enterocolitis, is the commonest type of non-ulcerative bowel disturbance in tuberculosis. There is characteristically an associated hypo- or achlorhydria with a breakdown of the pepsin-hydrochloric acid digestion. Undigested meat, particularly connective tisue, is hurried out of the stomach and by hyperperistalsis into the distal ileum and colon. There it is attacked by putrefactive bacteria. Phenol bodies, amines and hydrogen sulphide result. These are the very toxic substances of Metchnikoff's and Lane's autointoxication and of ptomaine and other food poisonings. They are currently accused by Volhard of being the cause of true uremia. Proteolytic bacteria can exist only in an alkaline medium. The ammonia produced by them is responsible for much of the alkalinity. The syndrome of putrefactive dyspepsia presupposes a bowel capable of irritation by the products of proteolysis. In the colon such substances are normally represented by indole, skatole and cadaverine in low concentration. Abnormal reactivity of the bowel to proteolytic products or the tendency of protein-splitting bacteria to flourish may be constitutionally[5] determined or may be conditioned by the products of tuberculous inflammation.

The stool in putrefactive dyspepsia is loose, alkaline and has a very foul odor. It is usually dark green. Pieces of muscle may be found microscopically.

Fermentative dyspepsia and fermentative enterocolitis are basically due to failure of carbohydrate digestion and absorption. Cellulose, the starch more resistant to physical and chemical disintegration, is the food component most concerned. Entering the colon undissolved, the starch is split by bacteria into lower fatty acids and aldehydes irritating to the bowel wall. Such bacterial action is a normal, necessary process in grazing animals.

The neurogenic factor is important in the production of fermentative as well as of putrefactive dyspepsia. The threshold of stimulation to the acid products of fermentation varies widely between individuals and from time to time in the same individual. The concentration of acid substances or of mechanically irritating pieces of cellulose causing marked symptoms in one person may be entirely unnoticed by another. Pain and tenderness are intermittent or continuous here as in putrefactive dyspepsia. Symptoms peculiar to the fermentative type are distension and marked flatus. Evacuations in fermentative dyspepsia are frequent and explosive. They are often accompanied by burning at the anus. The stools are light-colored and frothy or watery. They have an aromatic odor and an acid reaction. Microscopically, starch is recognizable on adding iodine.

Another less clearly defined but important group of tuberculous patients with intestinal complaints are those with predominating nervous symptoms. In them diarrhea may alternate with constipation. The stools are usually neutral to litmus. Periods of increased fermentation occur. Laboratory and x-ray signs of ulcer-

ation are lacking. This group is well characterized by the title of "irritable bowel."

Mucous colitis is a diagnosis often given to patients with symptoms of irritable bowel whose stools contain notable amounts of mucus. Mucus is a normal secretion of the intestinal glands. It is produced in excess in response to any irritation of the bowel. It may be a nervous reaction expressed as stimulation of the intestinal glands. Mucous colitis is a loose term.

The harmful effects of either structural or functional bowel disease in tuberculosis are far-reaching. The discomfort and the frequent defecations interfere with relaxation in the day and with sleep at night. Diarrhea results in defective absorption of proteins and of the calorigenic carbohydrates and fats. The loss of salts in diarrheal stools is particularly undesirable in a chronic illness with a tendency to demineralization. The development and maintenance of resistance to the infectious process is unfavorably affected by decreased absorption, inappetence and a fear of eating "because everything runs right through."

Prevention of gastro-intestinal complications in pulmonary tuberculosis consists primarily of instruction in nutrition and in the hygiene of eating. Patients should come to meals rested and unhurried. They should eat leisurely. Slow-eating may be furthered by serving in courses. The meals should be attractively prepared. Many of the patients have been "picky" eaters all their lives. They have never known proper eating discipline. Uncontrolled in dining rooms or by not checking trays, they choose very unbalanced meals and meals often very deficient calorigenically. They carefully cut the fat from their meat and the crust from their bread. I feel that these long-standing eating habits are causal in the tuberculosis and not always to be condoned with the excuse of tuberculous anorexia. Patients should know that cigarettes before meals raise the blood sugar high enough to decrease appetite and in some people provoke hyperperistalsis and hypersecretion, undesirable results in the syndromes under consideration. Adequate doses of vitamin B should be persistently given, not used in experiments of two weeks. Cowgill's work has shown that our dietary needs doses far in excess of those usually administered. The doses must be especially high in the presence of infection. Vitamin D should be offered in some feasible form. The currently used oils, cod and halibut, contain also high concentrations of the A vitamin. McConkey has emphasized the importance of vitamin C. If fresh vegetables and fruits are not tolerated, cevitamic acid may be given. Both B and G are other necessary components of special importance in bowel physiology. Overdosage with these vitamins is unknown, but yeast and wheat embryo cause diarrhea in some individuals. Such an effect is of course to be shunned.

The control of the tuberculosis in the lungs is the fundamental necessity for the control of its complications. The patient should be repeatedly cautioned against swallowing sputum.

Of the general and local measures directed at the gastro-intestinal complications, rest is of primary importance, both in prevention and relief. In many instances strong tea and coffee should be prohibited. The time and subjects of radio programs should be controlled. It should be borne in mind that this class of patients tends to excess in these particulars. Progressive relaxation[4] in Jacobson's sense is the most practical formal mental therapy for this group.

Heat is the most useful and easily applied physical agent. It may be used as the hot water bottle or electric pad, but most effectively in the form of the hot pack. The heat lamp is a clean, easily applied method of heating the abdomen. The patient must be kept warm. He should avoid bathing in cold water.

Ultra-violet and x-rays have been extensively used in the treatment of intestinal tuberculosis. They are of questionable benefit.

Of food adjuncts, such minerals as calcium are indicated because the demineralizing process of the tuberculosis is accelerated by diarrhea. Calcium also tends to neutrality in the intestinal lumen. Calcium salts make for nervous stability. Vitamin D is for the absorption of calcium. Hardt has recently re-emphasized the usefulness of calcium salts in intestinal tuberculosis.[5]

The gastric factor is frequently underestimated. We cannot approximate by the oral route the quantities of hydrochloric acid secreted by the stomach. In spite of this, the administration of hydrochloric acid sufficiently diluted and in increasing doses often brings about prompt cessation of symptoms in instances of hypo- or achlorhydria. Most patients can eventually take

6 cc. of acid hydrochl, dil. (U.S.P.) three times daily. I have them mix it in two glasses (400 cc.) to take one-third before, one-third during, and one-third after meals. In this dilution it does not injure the teeth. It often increases the appetite.

Belladonna to relax spasm and bromides or barbital to lower the nervous irritability are the most useful drugs. Opium may be used with benefit. Antiseptics are useless: Laxatives or large enemas are contraindicated. Small enemas (100 cc.) may be used in constipation but not laxatives.

Pneumoperitoneum has recently been advocated.

In general, abstinence from irritating substances such as condiments, salad dressing, pickles and cold food are necessary. Vegetables and fruits should be cooked and, possibly be pureed until the patient's gastro-intestinal tract has become very tolerant. Food should not be fried. The old process of "hanging" meat permitted autolysis to make connective tissue more digestible.

The management of fermentative and putrefactive dyspepsia is identical with that in non-tuberculous conditions. Adolf Schmidt starts his régime with a mixed, bland diet during the diagnostic period.

A starvation period of two to four days may precede the test diet if diarrhea is present. During starvation only 500 cc. of weak tea or a mixture of hot water and cognac is given by mouth. Acetonuria and fluid depletion are avoided by dextrose solution hypodermically or intravenously.

Of course, the patient must be confined to bed during this test period.

The *test diet* follows:

On arising—500 cc. milk, tea or cocoa. Biscuit of white bread. Butter. Soft-boiled egg.

9:30 A.M.—Plate of sieved oatmeal broth with salt or sugar.

Noon—One-fourth pound of finely chopped lean beef broiled, rare. Butter added after cooking. Generous helping of sieved potato broth.

3:00 P.M.—Tea or cocoa with a biscuit and butter, or 500 cc. of milk.

Supper—Plate of meat broth, thickened with flour, or 500 cc. of milk. Biscuit with butter. One or two soft-boiled eggs.

The order of meals may be changed. Bouillon may be added. Veal may be substituted for beef. Meat should be omitted during examination for occult blood. At the end of two or three days examination of the stools will have indicated the type of dyspepsia.

The general management of *putrefactive* dyspepsia calls for the elimination of meat, eggs and foods mechanically stimulating secretion and peristalsis as rough meal, coarse vegetables and vegetables with hulls as corn, peas and the granules of potato. Such chemical irritants as fruit juices, coffee, salty foods and drinks, cold drinks, charged water, extracts of meat including strong meat broth and gravy, yeast, spices and fried food are also to be avoided. Fats are well tolerated except in cases complicated by gallbladder disease or amyloidosis.

A preliminary fast of one to three days is desirable to rid the colon of protein derivatives. Give water or water flavored with rice or oatmeal. The patient should rinse his mouth with hydrogen peroxide. Glucose should be given by needle.

Second Stage

150 cc. to 300 cc. of ten per cent. cane sugar in water or weak tea five to seven times daily. Continue for three to four days.

Third Stage

Gradually replace the sugar water with boiled milk, 1,500 cc.-2,000 cc. daily. After a week, gradually add warm, cooked cream up to 300 cc. Add fresh cottage cheese during this second week of management. Slowly replace the milk by smooth soups and broths of cereal (baby food, sago, tapioca, arrowroot). Feed every two hours. Allow cognac or rum diluted ten times with water.

a. If milk is not tolerated, give 50-g. to 80-g. toasted white bread five to seven times daily. Give fluid only in sips. Chew slowly. One liter dextrose solution intravenously.

b. Instead of toast, 40 g.-50 g. well cooked rice is given dry five to seven times daily. Dextrose is given intravenously. Chew slowly to induce a maximal secretion of saliva.

This third stage should be continued for one to two weeks.

Fourth Stage

a. Meat-free. The same as the third stage including the modifications.

On arising, tea—milk (one to two teaspoonfuls

of tea leaves in 250 cc. of hot milk. Toast or zwieback with butter.

10:00 A.M.—General gruel. Toast and butter.

Noon—Cereal soup. Rice. Toast. Butter. Cottage cheese.

3:00 P.M.—300 cc. boiled milk. Toast and butter.

Supper—Like noon.

Bed-time—300 cc. boiled milk.

b. Add one to two eggs. Gradually substitute leguminous (powdered strained) meal for soup.

c. Add ground beef (well cooked), breast of fowl or fish. Jello. Pureed carrots, artichokes, young peas, spinach, asparagus.

The management usually requires two to five weeks. Return to normal diet gradually. Remember that connective tissue is the worst offender in putrefactive dyspepsia. Where indicated, supply pancreatin in addition to pepsin and hydrochloric acid.

In fermentative dyspepsia fermentable material must not enter the lower ileum or colon. Failures in management are attributable to a lack of carbohydrate restriction or to limiting carbohydrates for too short a time. Six to eight days are required to change the bowel flora by starving out the fermenting bacteria.

Start with fluids. Then give pulverized food and finally coarser food. Eat slowly. Chew thoroughly.

Fasting for one to two days is desirable.

In the mild cases give 150 cc. of weak tea (unsweetened or saccharinized) every two or three hours.

In more severe cases give all fluid by needle.

First Diet

On arising—300 cc. of albumin milk. May be heated and flavored with tea leaves. Two soft eggs.

10:00 A.M.—200 g. fresh cottage cheese. Enrich with 40 g. of butter after a few days.

Noon—Meat broth (beef tea or cooked bone marrow) or jellied bouillon. Soft, well boiled or roasted meat or fish. Chop meat for poor chewers. Gelatin sweetened with saccharin. Butter.

3:00 P.M.—400 cc. albumin milk. Soft-boiled egg.

Night—Buttered eggs and soft boiled ham. 40 g. old Holland cheese or cottage cheese with butter. Thin tea flavored with rum, if desired.

Continue this diet six to ten days or until starch disappears from the stool and the stool becomes alkaline. In case of relapse, return to this diet.

Second Diet

Increase the quantities. Add powdered rice, sago, tapioca, arrow root or corn starch. Give cooked fruit juices. These substances will be absorbed in the upper small intestine.

In severe cases, follow the second diet with three days of albumin milk, toasted white bread, butter and cottage cheese.

Third Diet

Gradually replace the albumin milk with whole milk up to 1,500 cc. daily. White bread with plenty of butter. Finely pureed young carrots, spinach, kohl-rabi, asparagus tips. Later, potato broth and strained apple sauce. Rice pudding, zwieback and toast. Fresh, soft noodles, but no other doughy food. Light desserts of rice or white wheat flour with egg, sugar and cream flavored with vanilla. Chocolate and cocoa. Can give fat beef and fat ham. No fried food.

On arising—Tea or cocoa with milk. White bread. Butter. Two soft-boiled eggs. Ham cooked until it is soft.

10:00 A.M.—400 cc. of albumin or whole milk. White bread. Butter.

Noon—Meat broth. May add rice or white flour to it. Tender meat or fish. Potato broth. Pureed vegetables. White bread. Butter. Mild cheese. Light pudding, replaced later by apple sauce.

3:00 P.M.—Milk or albumin milk. White bread. Butter.

Night—Soft eggs. Meat or fish. White bread. Butter. Cottage cheese.

Bed-time—300 cc. of milk.

Fourth Diet

Period of acclimatization to cellulose and to raw starch. Use coarser pureed food for a time. Keep under observation.

Calcium carbonate can be given to reduce the acidity of the feces.

The outcome of efforts to restore normalcy to the gastro-intestinal tract depends first of all upon the extent of the pulmonary and intestinal pathology. Ulcers involving the muscularis are certain to scar in healing. According to the general assumption there is little hope for patients with tuberculous ulceration with diarrhea. In one of their commonest locations, the small

bowel, ulcers are often undiagnosable by x-ray. Consequently their frequency in patients going on to recovery is unknown. The autopsy percentages of 50 per cent. to 93 per cent. of gastro-intestinal ulceration in pulmonary tuberculosis is of little help in estimating the frequency of ulcers in clinical material or the likelihood of ulceration in a given patient. Friedberg has frequently found scars of healed intestinal ulcers. Brown and Sampson[2] report good results from heliotherapy and from McConkey's cod liver oil and vitamin C feeding. Certainly in the gastro-intestinal tract, as elsewhere, the great tendency for tuberculosis to heal should be fostered. One of the greatest obstacles is the tendency of the patient to take a laissez-faire attitude, leaving everything to his medical attendant, the patient himself making little or no effort to render such assistance as eating necessarily monotonous food and resting faithfully.

Other prognostic factors are constitutional and hence outside the control of doctor or patient. The patient with the phthisical or asthenic habitus is predisposed by intestinal ptosis to intestinal disease. Nissl and von Noorden have emphasized the importance of the individual's strain of colon bacilli. Luxuriantly growing colon bacilli protect the bowel by overgrowing organisms passing the gastric barrier. A certain constitutional inferiority of the intestinal tract must be postulated for some individuals and indeed for whole families.[6] This may simply be a part of the general status degenerativus of which the tuberculosis is another expression. People with constitutional weakness of the intestinal tract will more easily develop ulceration and show greater irritability of the nervous mechanism of the bowel than will those without such a constitutional taint.

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THE TREATMENT OF GONORRHEA WITH A NEW SULFANILAMIDE DERIVATIVE

GERALD C. HUNT, M.D.
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THE STUDY

The study comprises a series of thirty-five cases of gonorrheal urinary infection, encountered in private practice, and treated with a new sulfanilamide derivative, sulfanilyl-sulfanilamide, manufactured under the name of disulon.

DOSAGE

The dosage consisted of 45 grains per day for two days followed by 30 grains per day for seven days. Only one course was given in most cases. In one case (26) a smaller daily dose was given because of the large previous therapy. Two courses were given in case 15 with success and with no untoward effects.

TEST OF CURE

A cure was substantiated by repeated smears, by clinical observation, by urinary examination, and finally by the gonorrhea-fixation test of the blood. In my set-up there were no provisions for culture, or for determination of drug concentration in the blood, urine and feces.

SEX AND INCIDENCE

This series consisted of thirty-four males and one female, ranging in age from 17 to 42, with an average age of 23.4 years. Seventeen cases were purely acute to the use of disulon; the remaining cases were preceded by courses of sulfanilamide.

ANALYSIS OF CASES

Cases of specific urethritis treated with disulon alone—9 cases (25.7%).

Cases of specific urethritis treated with disulon and local medication—10 cases (28.6%).

Cases of specific urethritis previously treated with sulfanilamide—16 cases (45.7%).

RESULTS OF CASES

	Cures	Failures
Disulon used alone.....	7	2 (22.0%)
Disulon and local medications.	9	1 (10.0%)
Sulfanilamide and disulon....	13	3 (18.7%)
	29 (82.85%)	6 (17.14%)

No.	Initial	Age	Sex	Smear	Diagnosis	Duration of Symptoms	Treatment	Length of Cure	Remarks
1.	C. C.	25	M	Pos.	Acute Anterior Urethritis	4 days	Disulon Alone	9 days	After 3 days no discharge
2.	H. C.	35	M	Pos.	Acute Anterior Urethritis	8 days	Disulon Alone	9 days	After 5 days no discharge
3.	G. M.	28	M	Pos.	Acute Anterior Urethritis	1 day	Disulon Alone	9 days	After 3 days no discharge
4.	P. M.	41	M	Pos.	Acute Anterior Urethritis	6 weeks	Disulon Alone	12 days	After 2 days no burning; after 6 days no discharge
5.	E. T.	24	M	Pos.	Acute Anterior Urethritis	8 days	Disulon Alone	11 days	After 2 days no discharge
6.	L. T.	23	M	Pos.	Acute Anterior Urethritis	6 days	Disulon Alone	7 days	After 2 days no discharge
7.	B. Y.	21	M	Pos.	Acute Anterior Urethritis	2 days	Disulon Alone	7 days	After 2 days no discharge
8.	T. B.	27	M	Pos.	Acute Anterior Urethritis	4 days	Disulon and Local Treatment	7 days	No discharge on following day
9.	J. A.	39	M	Pos.	Chronic Anterior Urethritis	3 weeks	Disulon and Local Treatment	11 days	No signs or symptoms after 3 days; few shreds in urine
10.	S. C.	22	M	Pos.	Acute Anterior Urethritis	1 day	Disulon and Local Treatment	4 days	No discharge on following day
11.	R. G.	29	M	Pos.	Acute Anterior Urethritis	4 days	Disulon and Local Treatment	5 days	No discharge on following day
12.	P. K.	17	M	Pos.	Acute Anterior Urethritis	1 day	Disulon and Local Treatment	9 days	No discharge on 4th day
13.	L. M.	31	M	Pos.	Acute Anterior Urethritis	1 day	Disulon and Local Treatment	10 days	No signs or symptoms after 3rd day; few shreds in urine until 10th day
14.	W. M.	21	M	Pos.	Acute Anterior Urethritis	4 days	Disulon and Local Treatment	4 days	No discharge on following day
15.	J. W.	39	M	Pos.	Acute Anterior Urethritis	3 days	Disulon and Local Treatment	4 days	First course of disulon stopped discharge, but glass No. 1 remained hazy; 2nd course of disulon, 1 mo. later gave a cure in four days
16.	W. E.	19	M	Pos.	Acute Anterior Urethritis	2 days	Disulon and Local Treatment	7 days	No discharge after 2nd day
17.	A. C.	26	M	Pos.	Urethritis Acute	4 days	Sulfanilamide and Local Treatment —Disulon	13 days	Two courses of sulfanilamide failed to check the discharge. 161 days preceded the use of disulon
18.	F. B.	22	M	Pos.	Acute Anterior Urethritis	3 days	Sulfanilamide and Local Treatment —Disulon	12 days	One course of sulfanilamide failed; no discharge after 3rd day under disulon 30 days preceded use of disulon
19.	F. H.	30	M	Pos.	Acute Anterior Urethritis	12 hrs.	Sulfanilamide and Local Treatment —Disulon	9 days	Under sulfanilamide there was an apparent cure until a relapse. The 2nd course of sulfanilamide caused cyanosis, dyspnea and severe backache. Under disulon discharge ceased within 2 days. 51 days preceded the use of disulon
20.	G. H.	19	M	Pos.	Acute Anterior Urethritis	2 days	Sulfanilamide and Local Treatment —Disulon	13 days	Sulfanilamide checked the discharge, but shreds remained in the urine. 15 days preceded the use of disulon
21.	J. J.	22	M	Pos.	Acute Anterior Urethritis	2 days	Sulfanilamide, Disulon	9 days	One course of sulfanilamide failed completely. 30 days preceded the use of disulon
22.	J. L.	23	M	Pos.	Acute Anterior Urethritis	3 days	Sulfanilamide, Disulon	10 days	Two courses of sulfanilamide failed completely; under disulon the discharge ceased in 3 days. 90 days preceded use of disulon
23.	R. P.	24	M	Pos.	Acute Anterior Urethritis	11 days	Sulfanilamide and Local Treatment —Disulon	11 days	Two courses of sulfanilamide failed; under disulon the morning drip ceased after 2 days. 62 days preceded the use of disulon
24.	D. S.	23	M	Pos.	Acute Anterior Urethritis	1 day	Sulfanilamide and Local Treatment —Disulon	11 days	Twenty-two days preceded the use of disulon
25.	E. S.	25	M	Pos.	Acute Anterior Urethritis	4 days	Sulfanilamide and Local Treatment —Disulon	10 days	Forty-two days preceded the use of disulon
26.	L. S.	26	M		Chronic Anterior Urethritis	1 year	Sulfanilamide and Local Treatment —Disulon	30 days	Three courses of sulfanilamide failed, as did the usual antiseptics, 151 days preceded use of disulon; the latter drug was used in small doses with success
27.	R. W.	42	M	Pos.	Acute Anterior Urethritis	3 days	Sulfanilamide and Local Treatment —Disulon	5 days	Three courses of sulfanilamide failed to clear up the urine, in fact there was a relapse. 5 mo. after onset, disulon was used with success
28.	I. D.	26	F	Pos.	Chronic G. C. Cervicitis Gonorrheal Arthritis	5 weeks	Sulfanilamide, Disulon	12 days	Under disulon all smears from cervix were negative after 12th day; disulon had no effect on the arthritis

No.	Initial	Age	Sex	Smear	Diagnosis	Duration of Symptoms	Treatment	Length of Cure	Remarks
29.	C. H.	32	M	Pos.	Chronic Urethritis Gonorrheal	3 weeks	Sulfanilamide, Disulon	11 days	Sulfanilamide failed in the specific urethritis. Disulon had no effect on the arthritis
30.	E. C.	24	M	Pos.	Arthritis	12 days	Sulfanilamide and Disulon Local Treatment	Failure	Sulfanilamide invoked a severe dermatitis; under disulon there was no change in the symptoms
31.	D. G.	33	M	Pos.	Acute Anterior Urethritis	10 days	Disulon and Local Treatment Sulfanilamide and Local Treatment	Failure	This patient became very cyanotic, necessitating reduction in dosage. A morning drop continued despite the treatment
32.	E. L.	17	M	Pos.	Acute Anterior Urethritis	3 days	Disulon	Failure	Sulfanilamide had no effect, disulon stopped the discharge but the urine remained cloudy. In this patient a diarrhea and urticaria developed on the 11th day under disulon. This disappeared on withdrawal of the drug, but reappeared when disulon was given the second time
33.	O. N.	35	M	Pos.	Acute Anterior Urethritis	3 days	Sulfanilamide Disulon	Failure	In this case disulon was given first with improvement but no cure; sulfanilamide likewise failed
34.	W. R.	23	M	Pos.	Acute Anterior Urethritis	2 days	Disulon and Sulfanilamide Local Treatment	Failure	Neither disulon nor sulfanilamide changed the course of this case
35.	C. S.	18	M	Pos.	Acute Anterior Urethritis	3 days	Sulfanilamide, Disulon and Local Treatment	Failure	The heavy discharge ceased under sulfanilamide, but the cloudy urine remained. Disulon made this patient very weak and nauseated

AVERAGE LENGTH OF CURE WITH DISULON

Disulon alone	9.14 days
Disulon and local medications	6.77 days
Sulfanilamide and disulon	12.00 days
Average	9.7 days

SIDE EFFECTS OR TOXIC REACTIONS

Nearly all the cases showed some minor effects of toxicity, including general weakness, cyanosis, nausea, dizziness and weakness. In case No. 19 there was a severe backache together with dyspnea and cyanosis. Cases No. 30 and 32 developed urticarial reactions; case No. 32, had a diarrhea. Case No. 31 became very cyanotic forcing a reduction in disulon dosage; case No. 35 became very weak and nauseated. In other words two cases developed dermatitis (5.7 per cent), one case diarrhea, whereas the remaining showed only minor symptoms.

CONCLUSIONS

1. It was observed that if the drug were to be successful it reacted almost immediately.

2. While using disulon, the happiness and confidence of the patient has been expressed favorably in the number of referred; other methods seem to place a "drag" on practice.

SUMMARY

1. This series presents 35 cases of acute and chronic gonorrhea treated with (disulon) sulfanilyl-sulfanilamide.

2. Twenty-nine or 82.85 per cent. were considered cured; the average length of time taking 9.7 days.

3. Sulfanilyl-sulfanilamide (disulon) had no effect on two cases of gonorrheal arthritis.

4. Two cases of dermatitis and one case of diarrhea were noted in this series.

5. The number of cases in this series are few, but there is an indication that disulon plus local treatment is more satisfactory than disulon alone and superior to sulfanilamide.

SOME TYPICAL CASE HISTORIES

No. 1, C. C., male, aged 25, presents a case which reacted uneventfully to sulfanilyl-sulfanilamide. On the first examination there was a profuse urethral discharge which began four days previously. The smear was positive, while glass number 2 was clear. He was placed on disulon immediately. Within three days all signs and symptoms had disappeared; within nine days both glasses were clear. The GC-fixation test and repeated smears were negative.

No. 15, J. W., male, aged 39, presents a case wherein a second course of disulon was given. This patient had a discharge of three days' duration. The smear was positive, and glass number 2 was clear. He was immediately placed on disulon. The discharge ceased within three days, but shreds were constantly present in the urine. A few days after disulon was stopped, a drop was present each morning, and these were positive to gonococci. For two months he was treated with various antiseptics, interspersed with rest periods, but without change. Again he was placed on disulon, and within four days all signs and symptoms disappeared, remaining so under repeated examinations.

No. 22, J. L., male aged 23, presents a case wherein sulfanilamide was used previously. This was a case of anterior urethritis with a discharge of three days' duration. The smear was positive, and glass number 2 was clear. He was placed under sulfanilamide immediately. No discharge was noted after the third day, but shreds remained in glass number 1. The latter remained during a rest period together with the reappearance of burning and a profuse discharge. He was given the second course of sulfanilamide with $KMnO_4$ locally. The only response was a decrease in the discharge. Three months after admittance he was placed on disulon. Within three days the discharge stopped completely, and within ten days the urine was clear. Repeated follow-up examinations were negative.

No. 28, I. D., woman, aged 26, presents a case of gonorrheal arthritis. This patient contracted gonorrhea from her husband five weeks prior to registration in this office. Two weeks later the right elbow became involved with the typical gonorrheal picture. Another physician had given her sulfanilamide, but her condition remained at a standstill. A cervical smear was positive to gonococci. She was allowed to finish the course of sulfanilamide, but the smear remained positive. She was then placed on disulon. The discharge ceased in four days, and the smear became negative on the 12th day, remaining so despite repeated examinations. During this time the arthritis showed little change. Later both sulfanilamide and disulon were tried again with no success on the arthritis. Other methods were resorted to with satisfactory results.

CASE HISTORIES

No. 34, W. R., man, aged 23, presents a failure to both disulon and sulfanilamide. The patient entered with a profuse urethral discharge of two days' duration. The smear was positive, and glass number 2 was clear. The disulon administration had no effect whatsoever on the discharge. Under sulfanilamide he was greatly relieved but a slight discharge remained. Later a second course of disulon stopped the discharge, and a cure was anticipated. One week later the discharge again became profuse, necessitating a change in treatment.

No. 32, E. L., man, aged 17, presents a case where a reaction followed the use of disulon. This patient was under the care of another physician who gave large doses of sulfanilamide with "arm injections" with complete failure. On examination the discharge was profuse, the smear was positive, and glass number 2 was clear. He was placed on disulon. On the fourth day the discharge had ceased. From the seventh to the eleventh day there was a morning drop. On the eleventh day he developed severe diarrhea with urticaria. With the removal of disulon, this disappeared in three days. On the twenty-first day he was placed on small doses of disulon. Six days later a very severe urticaria developed. Removal of the drug cleared up the condition. He was seen a month later and the discharge had returned, necessitating a different approach to treatment. This is listed as a failure because sufficient disulon was given to affect the gonorrhea if it were to act.

230 South Sixth Street.

MALIGNANT LESIONS OF THE CERVIX

CHARLES F. SHERWIN, M. D.
ST. LOUIS, MO.

The principal malignant conditions affecting the uterine cervix are carcinoma, sarcoma, endothelioma, perithelioma and, more rarely, corioma. Cervix malignancies comprise about eighty per cent. of all uterine cancers, and some writers say about twenty per cent. of all the malignancies occurring in women.

Of the various carcinomas the most common type is the squamous cell, which comprises about eighty-three per cent. of all cervix cancers. These originate in the vaginal surface epithelium and in the distal five or six mm. of the canal of the external os. The so-called acanthoma is a rare squamous cell carcinoma in which numerous epithelial pearls are found. The scirrhus carcinoma has much fibrous tissue about the squamous cells. The adenocarcinoma originates in the columnar epithelium farther up in the cervical canal and is much less frequent than the squamous cell variety. The cystadenocarcinoma results from the malignant degeneration of the mucous glands near the external os, and is comparatively rare.

The age incidence is more common in the fourth and fifth decades, although no decade is exempt. Ewing records a case of sarcoma of the cervix in a two-year-old child; cases of carcinoma appearing in the late twenties and early thirties are not uncommon.

The predisposing causes most often described are old cervical lacerations, repeated trauma from

Read at the sixty-fifth Annual Meeting of the Southern Illinois Medical Association, Nov. 2, 1939.

mechanical or chemical irritants, and cervical erosions. The last occurs especially where the columnar epithelium normally found in the upper two-thirds of the cervical canal becomes everted in to the vagina, where the mucous reaction is acid. Cancer developing in the cervix of a prolapsed uterus is very rare, thus some other factor than trauma by friction or mechanical means seems to be indicated. I strongly believe that endocrine (ovary or pituitary) hormonal factors play an important part in cervix cancer development. I might also suggest that inherited tendencies are too definite in many instances to be completely ignored.

The early symptoms are increased vaginal discharge, prolonged menstrual periods and, most important of all, spotting of blood after exercise or coitus. Pain is not an early symptom. Regular vaginal examinations twice a year are suggested as a valuable preventive measure.

The precancerous appearances are leukoplakia and small erosions or ulcers. These should be removed promptly when noticed, and the tissue microscopically examined. Conization of the cervix, trachelorrhaphy, removal of polypi, and curettement of the cervical canal when no external lesion is visible will all provide material for pathological examination, and will tend to prevent cancer development.

Far too many cases of erosions, cysts, old lacerations, small ulcers, or eversion of the columnar epithelium into the vagina are treated regularly and indefinitely by local caustics or tampons without any attempt at correction of the condition by simple excision or electrocoagulation. One often gets a history of several years of such "office gynecology" before the development of cancer, and frequently finds advanced cancer already subjected for several months to such trauma without even a biopsy having been taken. A small, sharp curette will easily remove several bits of diseased tissue for diagnosis. This can be done without an anesthetic, and is not considered a dangerous practice if the area is at once coagulated with phenol, nitric acid or the actual cautery. May I urge you to take biopsies of all erosions, ulcers or bleeding spots in the cervical canal, in order that cancer may be detected early and treated while still favorable? I also want to condemn the practice of supravaginal hysterectomy where a chronic cervicitis exists. I have seen three cases of cancer that developed in such cervices in the past year.

Schiller's iodine test, which consists of painting the cervix with weak Lugol's or Gram's iodine solution, has some value as a diagnostic sign. Cancer cells do not contain glycogen as do the squamous cells, and remain light colored in a darker brown zone. However, the columnar epithelium of the canal, if everted into an old laceration, will not stain as brown as the surrounding cells and is occasionally mistaken for actual cancer.

The prognosis in cancer of the cervix depends upon the degree of involvement and the promptness and thoroughness of treatment. The first degree is considered to be that of a small lesion limited to the cervix. The second group has a greater amount of cervix involved with the vaginal wall or body of the uterus invaded. Group three has definite extensions in the parametrium and broad ligaments. In the fourth group the bladder, rectum or abdominal nodes are also involved.

Treatment may be divided into prophylactic, curative and palliative. Let me stress again the great value to be derived by the repair of old cervical lacerations and the removal of ulcers, cysts, erosions and eversions of the columnar epithelium which appears much redder in color than the normal vaginal surface of the cervix. Always consider cancer a possibility, and do an early biopsy for diagnosis. If it is reported as cancer then treat it by one of the approved methods.

I prefer to use radium in the first and second groups, and occasionally as palliative treatment in the two advanced groups. When necessary I dilate the cervix wide enough to take the applicator easily, and remove any large masses of cancer if present with the electric cautery or high frequency scalpel. I then insert a T-shaped applicator with the stem in the cervix and the horizontal part transverse across the cervix, using 50 mgs. radium in a platinum capsule in each portion, which gives 1 mm. platinum filtration and 1 mm. rubber. Bladder and rectum are then packed off with a gauze strip placed firmly enough against the applicator to hold it in place, or else a suture is placed through the cervix and around the applicator. The radium should remain in place from forty-eight to sixty hours, giving a total radiation of from

4,800 to 6,000 mg. hours. If a lead capsule is used it should be about 2 mm. thick. I have not had a single slough into the bladder or rectum from such a technique, and seldom fail to see complete regression in an early cervical cancer where such dosage is used. The broad ligaments can later be treated by 10 to 20 mg. in a similar capsule with 3 mm. rubber on the ordinary spring colpostat, time forty-eight to sixty hours.

Radon implants of 1½ m.c. each to 1 cc. of tumor tissue are also valuable in thick areas or in the parametrium. Where definite bladder, rectal or broad ligament involvement is present, I refer the patient to the radiologist for deep-x-ray therapy in addition to the radium dose. I have one case of corioma involving the left side of the cervical canal well two years after cautery and 5,000 mg. hours of radium (patient of Dr. Andy Hall).

Nausea, which occasionally follows heavy radium or x-ray dosage, is usually controllable by intramuscular injections of liver extract and ten per cent. glucose solution given intravenously.

Being a firm believer in the one infallible rule in dealing with cancer, i.e., it never recurs in the tissue which the surgeon removes, I often suggest a hysterectomy following radium therapy. Even though the condition seems cured, occasionally nests of cancer cells may be devitalized, lay dormant for months, and later revive and grow again. If recurrences should appear, radium will not likely be as effective as it was in the primary instance. However, most patients decline an operation if they remain symptom-free. With parametrium involvement radium plus x-ray, followed by a Wertheim-type hysterectomy with removal of the lymphatic tissues along the ureters and iliac arteries, give the greatest chance of cure.

Palliative treatment by the actual cautery or by radium to control pain and hemorrhage and to heal ulceration has a justifiable place in cervix cancer management. Likewise, acetone coagulation of raw ulcerated areas is helpful to lessen hemorrhage and foul odors. I have sectioned the anterolateral tracts in the upper thoracic region of the spinal cord to control severe pelvic and low abdominal pain in a hopeless case. This is effective unless the metastases reach the pancreas level or along the aortic nodes above it.

Early cases treated by radium should give better than fifty per cent. five-year regressions. Those in the second stage may require surgery or x-ray in addition for about thirty per cent. favorable results, while the two more extensive groups will have only a small percentage of even one- or two-year regression periods.

IN CONCLUSION

1. Let me stress the preventive measure of cervix repair with the removal of destruction of all chronically inflamed, ulcerated, eroded or everted areas.

2. Consider the possibility of cancer in all such areas, and have an early biopsy examination.

3. If cancer is definite or strongly suspected, treat it with radium if early or with radium, x-ray and surgery if moderately advanced.

3720 Washington Blvd.

PLAGUE IN THE UNITED STATES

According to a survey made public today by the United States Public Health Service, there have been recorded in the United States 499 cases of plague in human beings, with 314 deaths, since the disease first made its appearance in this country in 1900. The first recorded appearance of plague in the United States, as well as on the North American continent, occurred in San Francisco, Calif, on March 6, 1900.

Just when or how plague was introduced into the United States is not known. Rot plague may exist in a city for some time without the development of the disease in human beings, and it is quite possible that the infection had been present among rats in San Francisco for many weeks or months prior to the discovery of the first human case, says Mr. Brock C. Hampton of the Division of Sanitary Reports and Statistics in the weekly publication of the Public Health Service (Public Health Reports Vol. 55, No. 26).

With only 8 cases of human plague reported in the United States during the 10-year period 1930-1939, the disease in human beings in this country may be thought to have become merely a matter of academic interest; but in view of the expanding areas in which plague-infection in wild rodents and insect parasites have been found in recent years, it assumes significant public health importance and becomes a problem fraught with potential danger.

Plague-infected rats were found in San Francisco during the first plague epidemic, and systematic efforts were made by the local health authorities, in cooperation with the Public Health Service, to destroy them, and to eliminate rat harborage, especially in the Chinese quarter. Notwithstanding such measures, the infection probably continued in these rodents in San Francisco and increased after the relaxation of suppressive measures to bring about a new human epidemic in 1907, following the earthquake and fire, which provided more

favorable conditions for the increase of rodents and fleas.

Since 1900, field investigations of plague and plague-suppressive measures have been conducted continuously, with varying degrees of intensity, in California by the State and local health authorities in cooperation with the United States Public Health Service; and since 1934 extensive field investigations have been conducted by the United States Public Health Service in cooperation with the health department of five western States. These studies have resulted in the discovery of wild-rodent plague in nine of the far western States, in addition to California, as follows: In Oregon and Montana in 1935; in Utah, Idaho, and Nevada in 1936; and in Washington State, Wyoming, New Mexico and Arizona in 1938.

To January 1, 1940, plague infection has been demonstrated in 14 species of ground squirrels, in red squirrels, tree squirrels, and flying squirrels, in wood rats, kangaroo rats, field mice, prairie dogs, chipmunks, marmots, and a cottontail rabbit in western United States, and in fleas, lice, and ticks from wild rodents. In May, 1939, plague infection was proved in a kangaroo rat trapped on April 15 about 10 miles west of Las Cruces, Dona Ana County, New Mexico. This is believed to be the farthest east and south that plague has been demonstrated in wild rodents in the United States, and the first instance of the proof of plague among kangaroo rats in this country.

Wild rodent plague has apparently been gradually extending eastward from the Pacific Coast. From the available evidence and the records of the Public Health Service it appears that plague infection has spread from the rats in San Francisco first to the ground squirrels and then to other wild rodents in western United States. It is possible that scavenger birds have played some part in spreading the infection. The burrowing owl, is a constant companion of the ground squirrel, frequently a joint tenant in its burrows. Fleas and ticks have been found in the nest of this owl, and other incriminating evidence has been found against it.

The spread of plague infection toward the East must be carefully watched; but with full knowledge available regarding the prevention and control of the disease, plague in epidemic form should never again be permitted to occur in any locality in the United States.

From the point of view of the clinical management of the individual case of tuberculosis and from the broader aspect of public health control of the disease, no one test occupies a position of greater importance and significance than that of the sputum examination. The persistence of a positive sputum is regarded as clear evidence that pathological activity of the disease has continued. An improved technic of sputum examination for acid-fast bacilli using tergitol has been reported. The use of this is said to approximate the results obtained by the use of guinea-pig inoculation, which is impractical except in selected cases because of cost. S. A. Petroff and P. Schain, Quar. Bull. of Sea View Hosp., January, 1940.

WAR . . . makes pleasant news for the tubercle bacillus. As the deaths from T.N.T. increase, those from tuberculosis lag not far behind. In the World War all countries showed this phenomenon whether under arms or not. What effect on our efforts to eradicate tuberculosis will these grim months ahead bring forth? . . . Unless we find a way to redouble the offensive against our hidden enemy, the sad story of twenty years ago will be told again and we will find ourselves facing a record of lost ground. Kendall Emerson, M.D.

There is no such thing as Mexican, White or Negro tuberculosis. The infection is passed from one race to another. No matter what his race, a person with tuberculosis is, first of all a human being, and, next a potential spreader of the trouble to others in the community. Health Crusader, April, 1940.

Marriages

EDWARD M. BURNS, Freeport, Ill., to Miss Pauline Elizabeth Hackett of Polo, June 18.

WILLIS W. BOWERS, JR., Granite City, Ill., to Miss Lois Eddy of Kenton, Ohio, June 1.

SAMUEL NIEDER, Gilman, Ill., to Miss Myrtle Levinson of Chicago, May 26.

ROLAND I. PRITIKIN, Chicago, to Miss Jeanne Du Pre Moore of Rockford, Ill., May 25.

LEONARD MARTIN THOMPSON, Lena, Ill., to Miss Esther M. Grier at Minneapolis, June 20.

Personals

Drs. J. P. Greenhill, Marshall Davison, George O'Brien and Herbert E. Schmitz motored to Fond du Lac, Wis., Thursday, June 27, to participate in the meeting of the Wisconsin State Medical Society at the Hotel Retlaw in Fond du Lac.

Dr. T. F. Reuther read a paper on the "Diagnosis and Treatment of Common Rectal Disorders" before the Macoupin County Medical Society, Carlinville, Illinois, June 4 and on "Nerves of the Anorectal Region" before the American Proctologic Society, Richmond, Virginia, on June 10.

Dr. Benjamin Boshes spoke before the Logan County Medical Society at Lincoln, Illinois, on June 27. His subject was "Recent Advances in the Study of Epilepsy."

Dr. H. Close Hesseltine participated in a program sponsored by the Hancock County Medical Society at Hamilton on July 8. His subject was "Use and Abuse of Sulfanilamide and Sulfapyridene in Gynecology and Obstetrics."

Dr. M. R. Guttman addressed the McHenry County Medical Society July 25 on the subject, "Diagnosis and Treatment of Middle Ear Complications."

Dr. Edward J. Stieglitz has resigned as a member of the staff of Presbyterian Hospital to accept an appointment at the National Institute of Health, Washington, D. C., where he will conduct clinical and experimental research on the problems of aging.

Dr. James B. Herrick was awarded the honorary degree of doctor of science at the convocation of Northwestern University, June 16, and in May was presented with the "Gold Headed Cane" by the University of California Medical School, San Francisco, for his accomplishments in the practice of medicine, in teaching and in investigation.

Dr. Ludvig Hektoen, professor emeritus of pathology, University of Chicago School of Medicine and Rush Medical College, was awarded the honorary degree of doctor of science by the university, June 11, "in recognition of his scholarly achievements in basic science and his distinguished service to the university and to medicine as a great teacher, editor and administrator." Dr. Hektoen since 1937 has been executive director of the National Advisory Cancer Council of the U. S. Public Health Service.

Dr. James P. Simonds has been named president-elect of the Chicago Medical Society and Dr. Frank F. Maple installed as president of the society. The ninetieth annual meeting and dinner of the society, June 25, was addressed by the outgoing president, Dr. Nathan S. Davis III, and by Dr. William Allen Pusey on "The First Ninety Years of the Society."

News of State

—At the annual meeting of the Chicago Gynecological Society held June 21, the following officers were elected: President, Harold K. Gibson, M. D.; President-elect, Charles E. Galloway, M. D.; Vice-president, Edward Allen, M. D.; Treasurer, Ralph A. Reis, M. D.; Secretary, James A. Gough, M. D., 104 S. Michigan Avenue, Chicago, Illinois; Pathologist, William J. Dieckmann, M. D.; Editor, Garwood C. Richardson, M. D.

—The Chicago Urological Society elected the following officers, May 22: William J. Baker, president; Norris J. Heckel, vice-president; Irving J. Shapiro, secretary-treasurer.

—The Educational Committee is receiving many requests for assistance with health programs before various types of lay organizations. Women's Clubs, Parent Teacher Associations, Home Bureaus, men's clubs have sent in requests from all sections of the state.

—The Chicago Society of Internal Medicine elected the following officers for the coming year: President, Clarence F. G. Brown; Vice-president, Robert Bloch; Secretary-Treasurer, Richard Capps.

—The staff of the Illinois Eye and Ear Infirmary announces a six months' course for orthoptic technicians, to start on October 1, 1940. The work will follow closely the recommendations of the orthoptic council. Applications should be accompanied by a letter of recommendation from an ophthalmologist and be sent to the Dean of Instruction, the Illinois Eye and Ear Infirmary, 904 West Adams Street, Chicago.

—Dr. Preston Kyes, since 1918 professor of preventive medicine, School of Medicine of the University of Chicago, became professor emeritus July 1. Dr. Kyes was born at North Jay, Maine, in 1875. He graduated at Johns Hopkins University School of Medicine, Baltimore, in 1900. He was a fellow of the Rockefeller Institute for Medical Research, New York, from 1902 to 1905; associate in the Chicago Memorial Institute for Infectious Diseases, 1904-1909, and Royal Prussian Institute of Experimental Therapy at Frankfort-on-the-Main, Germany, 1901-1905. He has been associated with the University of Chicago since 1902.

—Dr. J. S. Templeton, Pinckneyville, President of the Ilinois State Medical Society, addressed the Macoupin County Medical Society at a dinner meeting in the Gillespie Country Club, Gillespie, Illinois, on Tuesday evening July 23rd. Doctor Templeton spoke on "Obstetrics in Country Practice." A large number of physicians with their wives were present.

—The second annual prize of $250 of the Chicago Surgical Society was given to Dr. Leon J. Aries for a paper entitled "Experimental Analysis of the Growth Pattern and Rates of Appositional and Longitudinal Growth in the Rat Femur" and Dr. Carl Ireneus Jr. for a paper on "Experimental Bile Pancreatitis with Special Reference to Regeneration (Recovery) and to the Toxicity of the Hemorrhagic Exudate." The papers were presented at the meeting of the society May 10. Dr. Aries graduated at the University of Illinois College of Medicine, Chicago, in 1932, and Dr. Ireneus at the University of Illinois College of Medicine in 1936.

—If a person becomes sick in Illinois and is unable to pay for necessary medical care, although otherwise self supporting, the county of residence may be required to compensate the attending physician for such services as have been previously authorized by the supervisor of the poor. To this effect was a recent decision of the Appellate Court of Illinois, fourth district, in the case of *Buckmaster et al.* v. *Effingham County,* 23 N. E. (2d) 747, 302 Ill. App. 353. The appellants in this case, a group of physicians, after being duly authorized, treated the minor child of a day laborer who was financially able to provide his family with all the necessities of life other than medical care. The court held that under the Illinois law the county must pay for the services rendered by these physicians.

—A. L. Bowen, state director of public welfare since 1933, was fined $1,000 and was ordered removed from office, July 11, for negligence in connection with a typhoid epidemic at Manteno State Hospital in 1939 in which more than sixty lives were lost. The sentence was imposed by Judge James V. Bartley, Joliet. Mr. Bowen has spent many years in state service, having been superintendent of charities from 1917 to 1921. In 1931 he was again appointed superintendent of charities and was made welfare director by Governor Horner in 1933. Dr. Ralph T. Hinton, managing officer of the Manteno hospital at the time of the epidemic, and Mrs. Lillian Williams, dietitian, were indicted at the same time as Bowen, but charges against them were dropped, it was said.

—The Illinois State Medical Society has created a permanent committee on archives to collect pictures and all possible data concerning the early physicians in every county of the state. This action is the result of a photographic display at the recent state medical meeting of about 1,500 pioneer physicians of Illinois together with short sketches showing some of the highlights of their practice. The exhibit was under the supervision of Dr. Carl E. Black, Jacksonville, who has been interested in such a collection for more than fifty years.

—The committee on the Elizabeth McCormick Child Research Grant of the Institute of Medicine of Chicago announces that it has the sum of $1,500 to be awarded to qualified investigators in the Chicago area for the aid of research in child welfare. Projects should in a broad sense be in the field of pediatrics, it was said. Applications by letter will be received up to October 15 and the award will be made soon after that date. Address all communications to Dr. John Favill, secretary of the committee, 122 South Michigan Avenue, Chicago.

Deaths

Frank Marvin Banker, Franklin Grove, Ill.; Rush Medical College, Chicago, 1892; for many years county coronor; aged 72; died, May 30, of myocarditis and diaphragmatic hernia.

Joseph M. Campbell, East Moline, Ill.; Missouri Medical College, St. Louis, 1880; formerly St. Clair County coroner and sheriff; aged 86; died, May 15, of uremia.

Albert L. Corcoran, Fellow, A. M. A., Peoria, Ill.; Northwestern University Medical School, Chicago, 1892; aged 70; died, June 3, in the Methodist Hospital of cerebral hemorrhage.

John W. Craig, Fellow, A. M. A., Chicago; College of Physicians and Surgeons, Keokuk, Iowa, 1881; an Affiliate Fellow of the American Medical Association; member of the Iowa State Medical Society; aged 83; died, May 6, of chronic myocarditis.

Emil Alexius Edlen, Moline, Ill.; University of Minnesota College of Medicine and Surgery, Minneapolis, 1892; member of the Illinois State Medical Society; at one time city physician; aged 80; formerly on the staffs of the Moline Public Hospital and the Lutheran Hospital, where he died, May 10, of uremia, myocarditis and arteriosclerosis.

C. W. Gilham, West Point, Ill.; Homeopathic Medical College of Missouri, St. Louis, 1897; aged 67; died, May 2, at St. Mary's Hospital, Quincy, of pneumonia.

Lucius Barnes Goodyear, Dixon, Ill.; Physio-Medical College of Indiana, Indianapolis, 1894; served during the World War; aged 76; died, May 1, of self-inflicted bullet wound.

CHARLES HAMILTON, Carlock, Ill.; College of Physicians and Surgeons, Chicago, 1895; on the staff of the Brokaw Hospital, Normal; aged 70; died, April 15, of bronchopneumonia, arteriosclerosis, cerebral thrombosis and uremia.

AUSTIN ALBERT HAYDEN, Fellow, A.M.A., Secretary of the Board of Trustees of the American Medical Association, a distinguished worker in the field of ophthalmology and otolaryngology, died in Chicago, July 10, of coronary thrombosis.

Dr. Hayden was born in Shullsburg, Wis., Oct. 15, 1881. His premedical education was received at Creighton University in Omaha and at the University of Chicago. He received his M.D. degree from Rush Medical College in 1904, served his internship in St. Elizabeth's Hospital and served also as intern in St. Anne's Hospital. He was instructor in ophthalmology and did graduate work at the New York Post-Graduate Medical School from 1906 to 1908, and then was instructor in ophthalmology at Rush from 1908 to 1916.

In his professional life Dr. Hayden was active in the work of many medical organizations. He was a fellow of the American College of Surgeons, a member of the of the Board of Trustees of the American Medical Asso- American Otological Society, a former president of the Chicago Laryngological and Otological Society, and of the Chicago Medical Society. He was also a member of the Chicago Ophthalmological Society, the American Academy of Ophthalmalogy and Otolaryngology, the American Laryngological, Rhinological and Otological Society, a former president of the American Association of Railway Surgeons and a member of the Institute of Medicine and the Society of Medical History of Chicago.

In more recent years he had given much attention to the problems of those who are hard of hearing and had served as president of the Chicago League for the Hard of Hearing and of the American Federation of Organizations for the Hard of Hearing. He had also contributed greatly to the development of hearing devices and recently published contributions on this subject both in scientific periodicals and in such publications for the layman as *Hygeia* and the *Reader's Digest*.

In the American Medical Association Dr. Hayden had served as treasurer from 1922 to 1933 and since 1933 he had been Secretary of the Board of Trustees. He was devoted to his work for organized medicine, giving much of his time also to the recently organized National Physicians' Committee, of which he was secretary.

At the meeting of the American Medical Association just held in New York he worked many hours during the day and night in his official capacities not only on the Board of Trustees and in the work of the House of Delegates but also in charge of entertainment during the session. He became ill following his return from the meeting and had been almost constantly in the hospital since that date. Dr. Hayden was known to his friends for his intense activity, his constant courtesy, his genial friendship and particularly his leadership in medical advancement. The debt of organized medicine to men of his type who give so freely of themselves for the common good cannot be too greatly recognized.

RALPH POOL GRIMM, Fellow, A.M.A., Farmington, Ill.; St. Louis College of Physicians and Surgeons, 1909; aged 54; was killed, June 23, in an automobile accident.

BENJAMIN CHASE GROUT, Berwyn, Ill.; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1905; aged 69; died, May 12, of coronary occlusion and arteriosclerosis.

ALEXANDER RODERICK MCDONALD, Fellow, A.M.A., Chicago Homeopathic Medical College, 1897; Rush Medical College, Chicago, 1898; at one time professor of physiology at the Hahnemann Medical College and Hospital; aged 77; on the staff of the West Suburban Hospital, Oak Park, Ill., where he died, May 3.

IRVING WAGNER METZ, Springfield, Ill.; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1900; member of the Illinois State Medical Society; aged 67; died, May 12, of cerebral hemorrhage, arteriosclerosis and hypertension.

CHARLES H. LOCKHART, Fellow, A.M.A.; Witt, Ill.; Missouri Medical College, St. Louis, 1898; on the staff of St. Francis Hospital, Litchfield; aged 68; died, May 17, of cerebral hemorrhage.

JOHN H. OLIVER, Fellow, A.M.A., Kewanee, Ill.; Beaumont Hospital Medical College, St. Louis, 1896; past president of the Henry County Medical Society; on the staff of the Kewanee Public Hospital; aged 69; died, May 16, in the Evanston (Ill.) Hospital of coronary occlusion and arteriosclerosis.

JAMES ORA PEARMAN, Mahomet, Ill.; Rush Medical College, Chicago, 1885; aged 80; died, May 4, in the Burnham City Hospital, Champaign, following an operation for prostatic
Burnham City Hospital, Champaign.

CHARLES CLIFTON SCOTT, Princeton, Ill.; Rush Medical College, Chicago, 1891; member of the Illinois State Medical Society; aged 73; died, April 3.

ISRAEL FRED STOWE, Basco, Ill.; Bennett College of Eclectic-Medicine and Surgery, Chicago, 1910; aged 65; died, May 5, of cerebral hemorrhage.

HARRY HYMEN TANZER, Fellow, A.M.A., East St. Louis, Ill.; St. Louis University School of Medicine, 1926; assistant in gynecology and obstetrics at his alma mater; on the staffs of Christian Welfare Hospital and St. Mary's Hospital; aged 40; died, June 10, in St. Mary's Hospital, St. Louis, of carcinoma of the intestine.

BERT TRIPPEER, Fellow, A.M.A., Dwight, Ill.; University of Louisville (Ky.) Medical Department, 1894; Barnes Medical College, St. Louis, 1895; aged 68; died, June 7, in the Grant Hospital, Chicago, of mesenteric thrombosis.

ROBERT A. WARE, Chicago; Meharry Medical College, Nashville, Tenn., 1910; aged 56; died, May 16, of a gunshot wound.